The Journal of Thoracic Surgery

VOL. 22 AUGUST, 1951 NO. 2

Original Communications

BRONCHOGRAPHY AS AN AID IN PLANNING SURGICAL TREATMENT OF PULMONARY TUBERCULOSIS

WITH A PRELIMINARY NOTE ON PULMONARY ANGIOGRAPHY

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A SERIES of lung specimens were studied by injecting radiopaque material into the bronchi and pulmonary arteries and comparing the roentgenographic findings with those of bronchograms in living patients. The interpretation of bronchograms on this basis has been an aid in planning the surgical treatment for certain cases of pulmonary tuberculosis. In this study, which was begun by one of us (J. G.) a little over eight years ago, the bronchial trees and pulmonary arterial system of removed lung specimens were injected with radiopaque material under fluoroscopic control. A variety of radiopaque materials such as bismuth and the iodides were tried, including watery solutions with and without acacia. It was finally decided that an iodized oily solution, as used in bronchography, was the most satisfactory for this purpose. The resultant bronchograms and arteriograms were then correlated with the pathologic changes. It was noted that where there were structural alterations of lung parenchyma due to pulmonary tuberculosis, there were also consistent changes in both the bronchograms and the pulmonary arteriograms.

The lesions present in the lungs of this series of cases are of two apparently distinct types. In the one type, of which Case 7 is an example, the lesion is essentially limited to the large or small bronchi or both, and the surrounding alveolar structure is normal, except for scattered small, isolated scars. The bronchial lesions consist usually of a thin zone of dense collagen deposition beneath the intact epithelium, in the location of the smooth muscle of the wall. The muscle fibers are largely obliterated. In some bronchi of cases showing this lesion, there is seen more or less extensive mucosal ulcera-

From the Ray Brook State Tuberculosis Hospital, Ray Brook, N. Y.

Read (by title) at the Thirtieth Annual Meeting of the American Association for Thoracic Surgery, Denver, Colorado, April 15-18, 1950.

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tion with epithelioid and caseous tubercles in the underlying tissue. Presumably, this indicates the mechanism by which the more common lesion of peribronchial fibrosis has developed.

In the other type, exemplified by Case 9, virtually the entire alveolar architecture is replaced by diffuse scarring with foci of caseation or cavity formation. Many of the smaller bronchi and pulmonary vessels are completely effaced. The remaining bronchi course through the wide diffuse scars, are tortuous and usually dilated, but sometimes narrowed. The mucosa is finely serrated. The larger pulmonary vessels associated with these bronchi are patent, but their smaller branches are obscured in the scars and caseous areas, as are the smaller bronchi.

Many lungs show both types of lesion; areas of diffuse scar tissue traversed by tortuous serrated bronchi; and in other areas, normal or emphysematous alveolar structure, with localized peribronchial lesions. Case 8 is an example of a case showing both types. In this instance a recent bronchogenic dissemination had occurred in the lower portion of the left upper lobe. The bronchi show chiefly epithelial ulceration. Some of the tubercles in the bronchial wall project into the lumen. Vascular changes are not apparent.

It appears that the peribronchial fibrosis is responsible for the poor arborization seen in the films of the injected lungs. The involved bronchi show slight serration of the lumen similar to that seen in the bronchograms. In some areas smaller bronchi and bronchioles are seen throughout the lung and are uniformly patent. The orifices of these channels from the larger bronchi are apparently sufficiently narrowed by the peribronchial fibrosis to inhibit the flow of the iodized oil. The generalized emphysema, and the trapping of air noted during life may perhaps be attributed to the same mechanism.

In the normal lung the pattern of the bronchial tree and pulmonary arterial system were similar and paralleled each other in an orderly fashion. There was seen the graceful, delicate arborization with progressive diminution in size of the branches of both systems (Cases 1, 2, and 3). Where the lung contained well-established foci of disease, whether small or very extensive, both bronchi and pulmonary arteries suffered in alteration or obliteration (Cases 4 and 5). The bronchographic patterns observed in these specimens were essentially of two types, and are exemplified in Cases 6, 7, 8, and 9. (1) In extensively damaged parenchyma the proximal larger bronchi were dilated and had irregular, serrated margins. They were somewhat crowded together and stopped rather abruptly so that there was no filling in the distal portion of the bronchial tree.* In these removed specimens cavities filled readily. The pulmonary arteriograms of the areas showing the above bronchial changes revealed a marked paucity of patent arteries. (2) In areas with relatively recent or less extensive disease, such as in bronchogenic spread, the abnormality of the bronchial pattern was less obvious. Some of the bronchioles did not fill at all, while others, even though they filled out to the periphery, were somewhat

*How this differs from bronchograms of nontuberculous pyogenic bronchiectasis is the subject of another communication.

narrowed, and their fine lacy branches were markedly decreased in number. The larger bronchi did or did not appear normal, depending upon the extent of parenchymal involvement. The injected pulmonary arteries of recent disease still showed patency in some areas, and in others obliteration was seen as in older diseased areas.

Following this study of removed lung specimens a variety of conditions was studied by bronchography (Cases 10 to 25) in patients known to have pulmonary

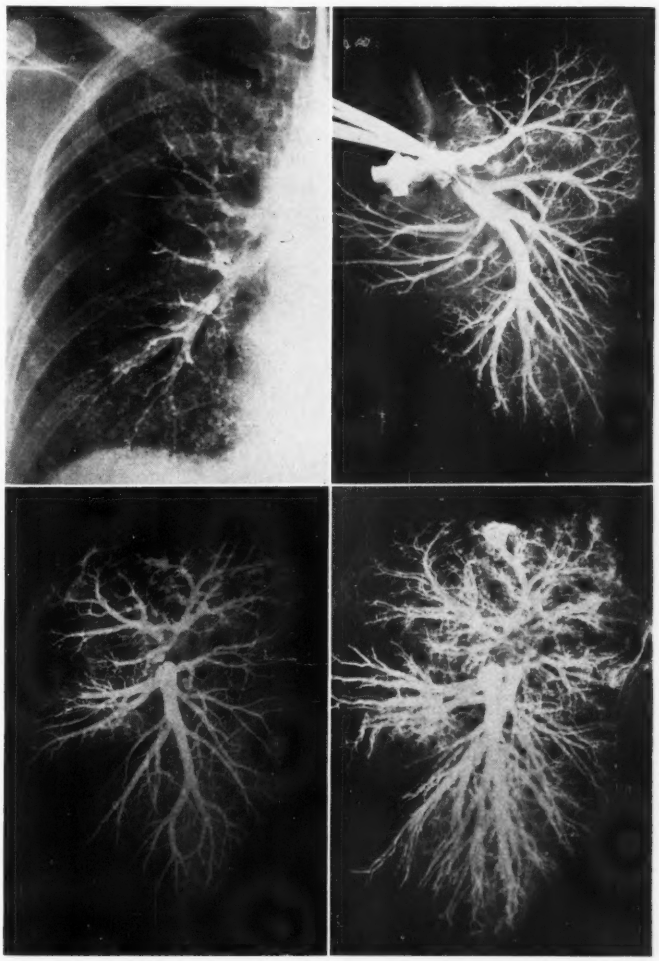


Fig. 1.—*1*, (*Case 1*) Normal posteroanterior bronchogram. *2*, (*Case 2*) Normal pulmonary arteriogram by injection of lung specimen. *3*, (*Case 3*) Normal pulmonary arteriogram by injection of lung specimen. *4*, (*Case 3*) Same lung with additional injection of bronchial tree showing superimposition of both patterns.

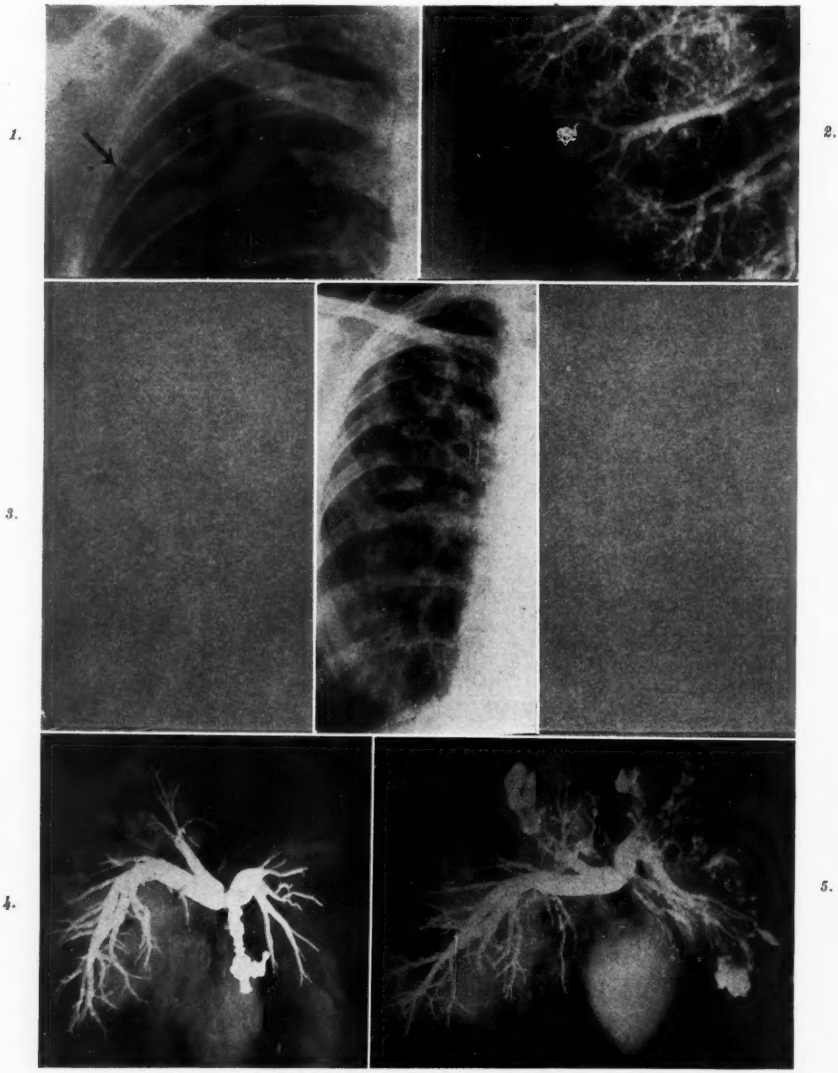


Fig. 2.—*1*, (*Case 4*) Posteroanterior roentgenogram showing shadow of small parenchymal tuberculous focus. *2*, (*Case 4*) Roentgenogram of injected lung specimen showing absence of filling of both pulmonary arterial branches and small bronchi in region of a small parenchymal lesion (by injection of both systems of lung specimen). *3*, (*Case 5*) Posteroanterior roentgenogram showing extensive changes on the right. Thoracoplasty on left not shown. *4*, (*Case 5*) Roentgenogram after pulmonary arterial injection of both lungs in postmortem heart-lung specimen showing marked paucity of arterial pattern. *5*, (*Case 5*) Same specimen with additional bronchial tree injection showing similar poor pattern and filling of cavities.

tuberculosis, and the following observations were made. The bronchographic pattern was altered in the presence of parenchymal disease, and again the two types of changes were commonly noted. With small individual areas of parenchymal involvement the only bronchographic changes were narrowing and poor

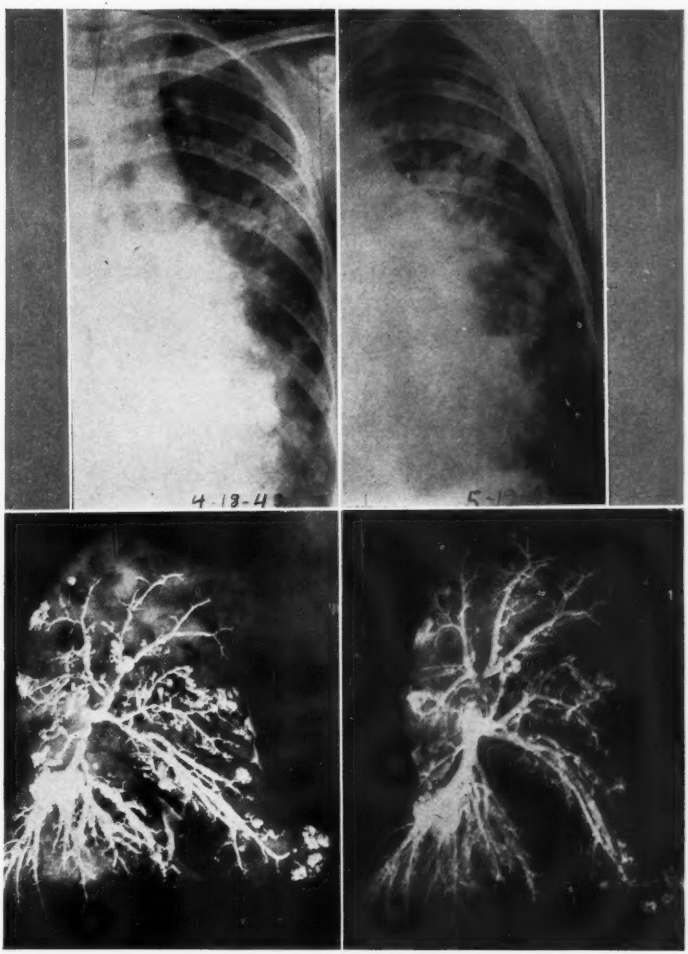


Fig. 3.—*1, (Case 6)* Posteroanterior roentgenogram, April 18, 1949, showing moderately extensive shadows on the left. *2, (Case 6)* Posteroanterior roentgenogram, May 19, 1949, showing new shadows of recent extension of disease. *3, (Case 6)* Roentgenogram after bronchial tree injection showing narrowing and lack of branching through diseased lung. At cavity which is filled the bronchus is short and wider than normal. *4, (Case 6)* Same specimen with addition of pulmonary arterial injection showing some relatively normal pattern in areas of recent disease and uninvolved parts of lung.

filling of some of the smaller bronchi (Cases 16, 18, and 19) so that normal arborization was lacking. With more extensive disease the distal portions of the bronchi did not fill at all, while the proximal larger bronchi were dilated (Cases 10, 11, 12, 13, 14, 15, 16, 19, 20, and 25), had irregular margins, and were

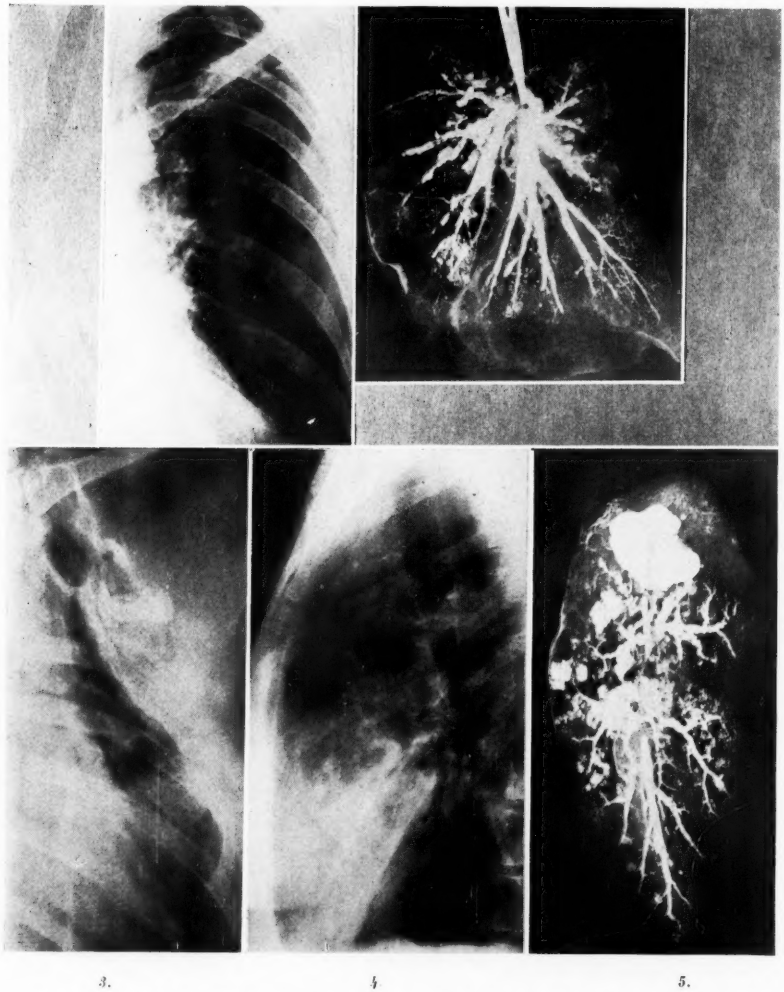


Fig. 4.—*1*, (*Case 7*) Posteroanterior roentgenogram of left lung showing few shadows. *2*, (*Case 7*) (Left pneumonectomy)—Roentgenogram after injection of both bronchial tree and pulmonary artery showing filling chiefly of larger divisions. (See pathologic description in Fig. 5.) *3*, (*Case 8*) Posteroanterior roentgenogram showing annular shadow—thoracoplasty failure—extension of disease into lower lung field. *4*, (*Case 8*) Left lateral roentgenogram showing location of disease in lingula. *5*, (*Case 8*) (Left upper lobectomy)—Roentgenogram after injection of bronchial tree showing only filling of cavity—bronchi to upper segment in region of cavity are short, wide, and have almost no branches, bronchi in lower segment showing narrowing and only few branches. (See pathologic description in Fig. 6.)

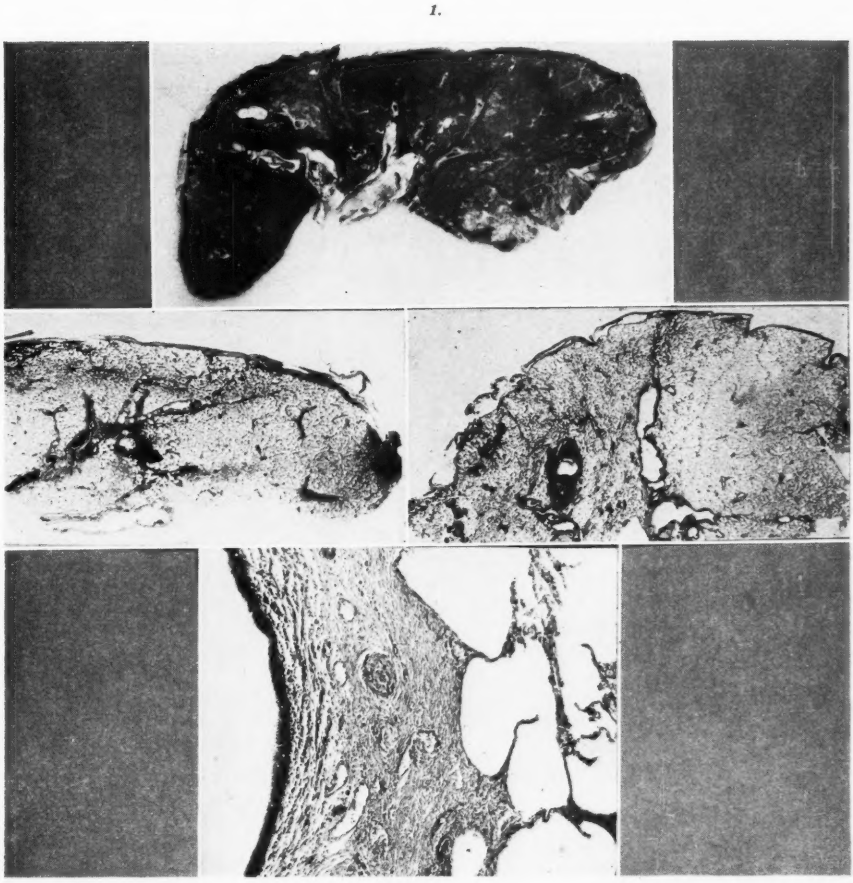


Fig. 5 (*Case* 7).—*1*, Gross photograph of left lung removed at operation. The upper lobe is at the left. Though rather few bronchi and vessels are seen, chiefly in cross section, it is apparent that all of them course through air containing pulmonary tissue. Note that the alveoli are generally coarse. The paucity of shadows in the chest roentgenograms is readily explainable by the appearance of the gross lung. The reasons for bronchographic changes, however, are less apparent. These changes depend upon the specific lesions associated with the bronchi, which are better shown in *2*, *3*, and *4*.

2, Low-power photograph of the histologic preparation from the lower lobe. Bronchi and vessels are seen extending into the lobe from the left. Their walls are thickened and their linings are irregular. The scattered minute round or elongated spaces with dark walls are bronchioles and arterioles which, though patent, were not outlined in the bronchograms. (Approximately ×1.5.)

3, Low-power photograph of the histologic preparation of the upper lobe of the lung shown in *1*. The oval dense focus is a dilated bronchus filled with exudate. At the center is a similarly dilated, but empty bronchus. At the left, beneath the pleura, are several small foci of encapsulated caseous material. In all areas there are minute patent bronchioles and arterioles seen in longitudinal and cross section. Generalized coarsening of the alveoli is apparent. (Approximately ×1.5.)

4, Photomicrograph of the wall of a bronchus which measured about 3 mm. in diameter in the section. The epithelium is intact, but the wall is greatly thickened by a dense concentric zone of scarring. The smooth muscle of the wall is obliterated. Dilatation and rupture (emphysema) of the adjacent alveoli is apparent. The contrast between this lesion and that shown in Case 8, Fig. 6, *4* is striking and apparently fundamental. (×70.)

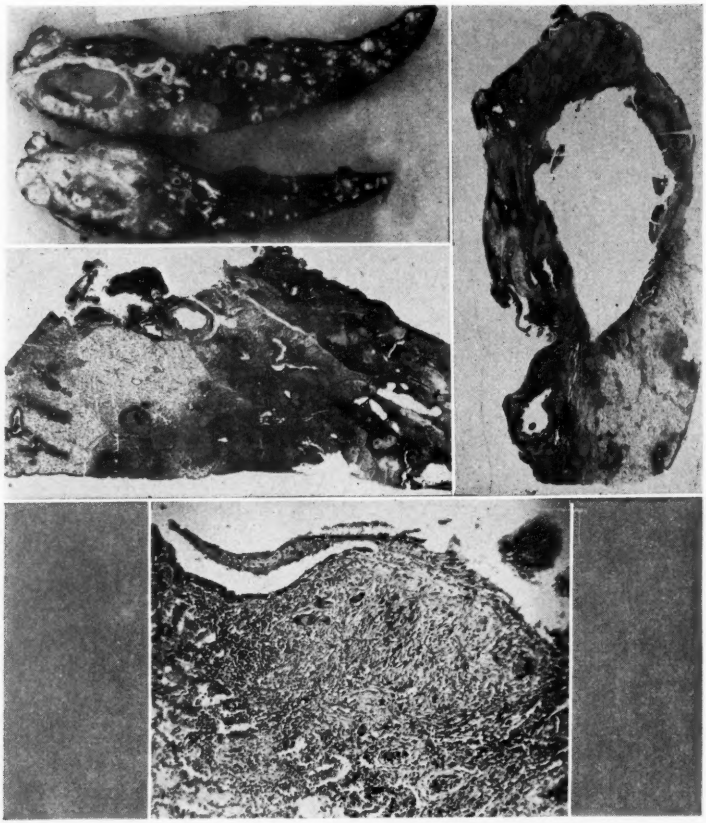


Fig. 6 (*Case 8*).— *1*, Gross photograph of two blocks of the left upper lobe removed at operation. The apex is at the left. The upper block shows a large cavity in the apical region. The lower block is more posterior and shows a rather extensive area of scar tissue and caseous material. The hilar bronchi show distinct thickening of their walls, and bronchi and vessels are largely obscured in the upper third of the lobe. In the lower portion of the lobe are seen numerous white foci of recently disseminated disease. Close inspection of these reveals that most of them are within or closely adjacent to small bronchi, the lumens of which can be identified. Close inspection also reveals many small vascular lumens, without associated foci of disease. These findings correlate with the roentgenogram studies which revealed poor filling of the bronchi, but good filling of the vascular tree. The dark areas are the result of hemorrhage into some of the secondary lobules during operation.

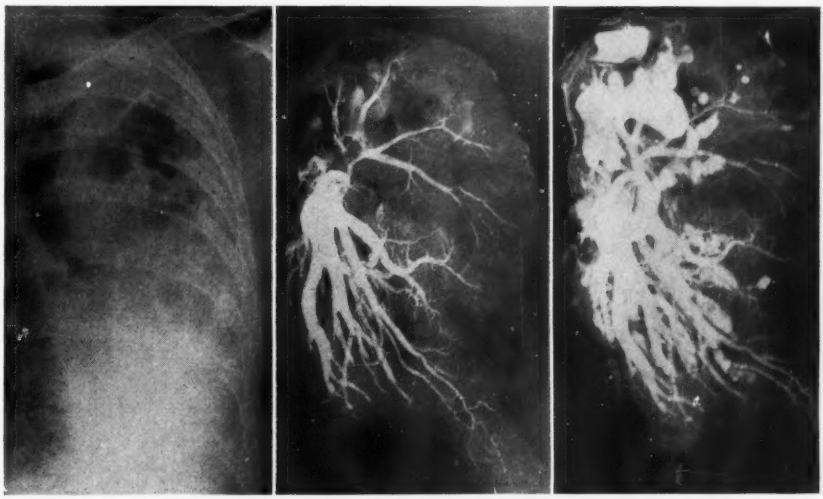
2, Low-power photograph of the histologic preparation from upper half of the lobe shown in *1*. In this section there are few bronchi, but the two adjacent to the lower border of the cavity show thickened walls and serrated lumens, like those in Case 7, (Fig. 5, *1*) (Approximately ×2.)

3, Low-power photograph of the histologic preparation from the lower half of the lobe shown in *1*. Operative hemorrhage obscures much of the field, and the lesions are best seen toward the left. Here several bronchi are seen in longitudinal section. Their walls are thickened and immediately adjacent alveolar tissue is consolidated. (Approximately ×2.)

4, Photomicrograph of the wall of a bronchus of similar size to that shown in Case 8, Fig. 5, *4*. The epithelium is ulcerated, but regenerating from the right, and a well-developed tubercle is protruding into the lumen. Adjacent alveoli are consolidated by exudate. It is likely that this is the early stage of the type of lesion which results finally in the peribronchial fibrosis seen in Case 7, Fig. 5, *4*.

crowded together.† The bronchograms in living patients did not appear to differ essentially from those of removed lung specimens, except that in the latter cavity filling was the rule rather than the exception.

Once these bronchial changes of chronic tuberculosis have occurred they appear to be irreversible, even though in some instances the diseased portion of the lung again becomes radiolucent as seen by routine chest roentgenograms. This has been observed, for example, in some cases of tuberculous pneumonia treated with streptomycin and P.A.S. Following the use of these drugs marked clearing of the dense shadows occurred leaving only linear strands. Bronchograms done one month to two years after this clearing showed an unanticipated extent of alteration in the bronchial pattern (Cases 17 and 20). In other instances this clearing has apparently been accompanied by marked resolution,



1. *2.* *3.*

Fig. 7 (*Case 9*).—*1,* Posteroanterior roentgenogram showing shadows of extensively destroyed left lung. Patient originally had cavity in lower lobe treated by pneumothorax. Ultimate re-expansion followed in few years by general breakdown of entire lung. *2,* (Left pneumonectomy)—Roentgenogram after injection of pulmonary arterial system indicating general paucity of pattern showing small lingular segment with some few branches, contracted lower lobe with similar absence of branching. *3,* Same specimen with addition of bronchial tree injection showing filled cavities and short wide bronchi with lack of branching. (See pathologic description specimen in Fig. 8.)

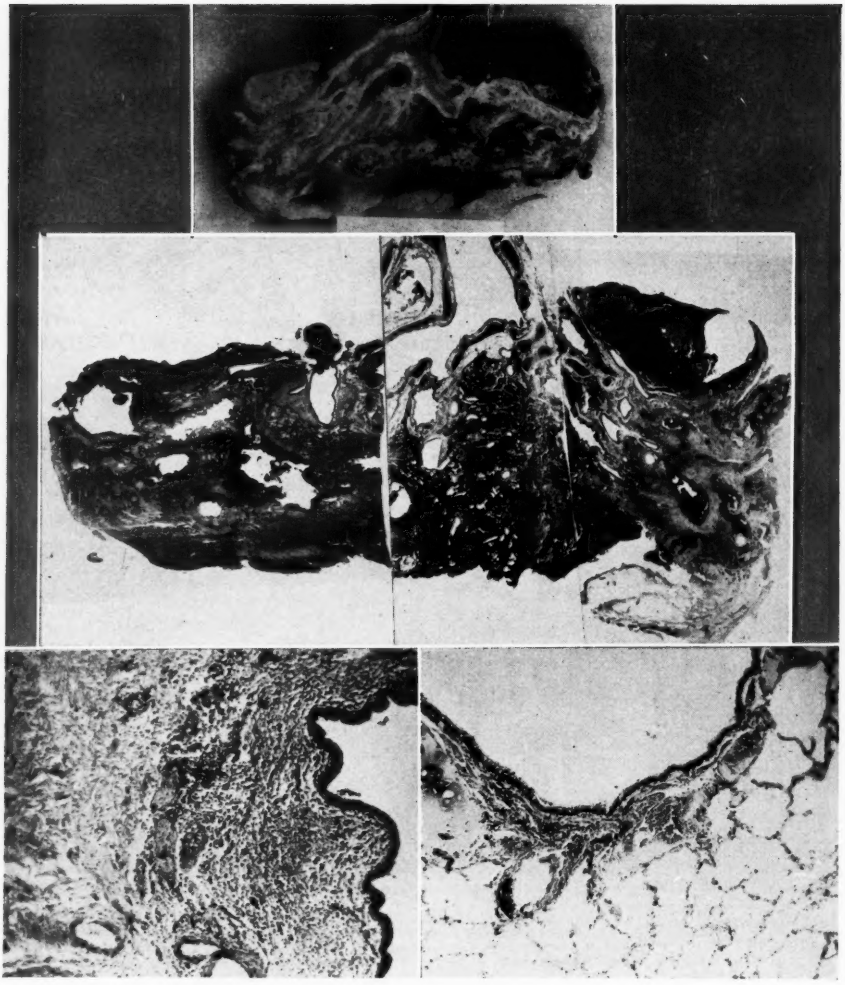
Case 9.—Pulmonary Function Study

Estimated vital capacity	5.02 L.
Determined vital capacity	3.85 L.
Estimated maximum breathing capacity	182 L.
Determined maximum breathing capacity	125 L.

By fluoroscopy the movement of the right diaphragm is within normal limits and the left diaphragm cannot be visualized. The right lung ventilates normally. The mediastinum is shifted moderately to the left on inspiration and returns toward the midline on expiration. There is little evidence of function on the left.

†The mechanism responsible for the bronchographic patterns is not clear and is under further investigation. It is thought that the latter finding may be due to partial collapse and retraction resulting from destruction of intervening parenchyma. The dynamics of the lung are such that the bronchi enlarge and elongate during inspiration and shorten and narrow in expiration. The damaged portion of the lung takes little or no part in normal respiratory function because of marked structural change including loss of elastic tissue. In this relatively collapsed or poorly functioning state the larger bronchi become fore-shortened and widened.

1.



2.

3.

4.

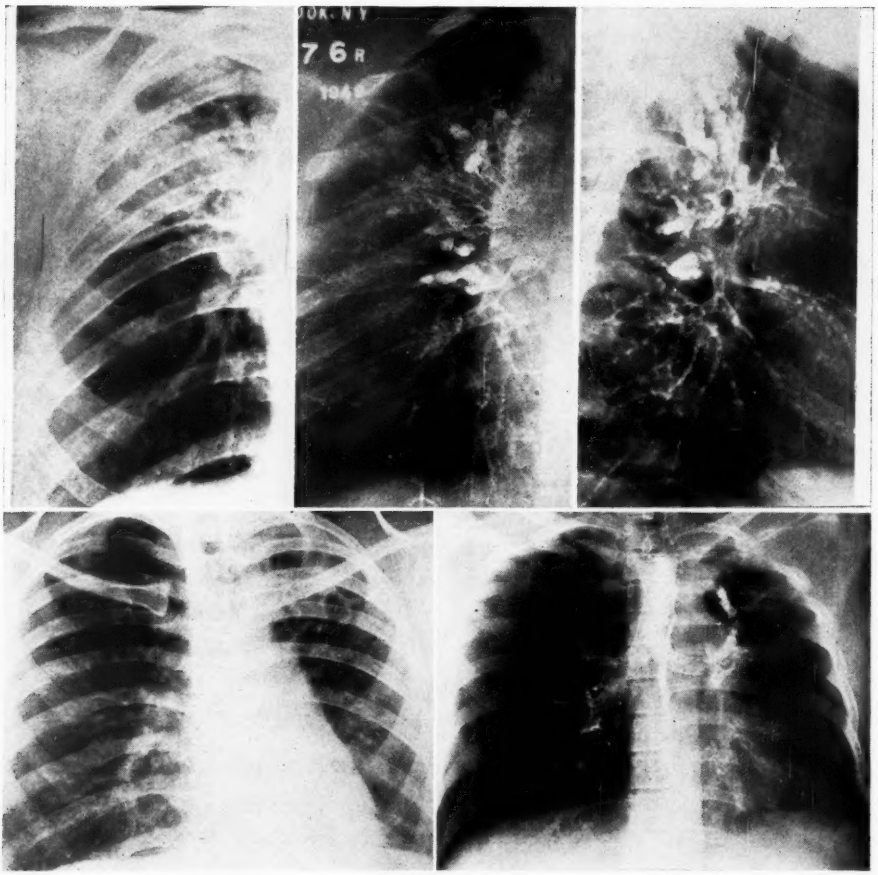
Fig. 8 (*Case 9*).—*1*, Gross photograph of the left lung removed at operation. The upper lobe is at the left, and the lower lobe is at the right. Two cavities are to be seen in the upper lobe, with a trabecula crossing the more lateral one. Between the two cavities a dilated tortuous bronchus is seen. It is this type of change, occurring in numerous bronchi, which produces the serrated appearance in the bronchograms. The same type of alteration is better seen in *2*, a composite photograph of the histologic preparations of this lung. The walls of the hilar bronchi are thickened, but their lumens are of essentially normal size. Hilar vessels, several of which may be seen between the main lobar bronchi, also show thickening of their walls, but essentially normal size. Smaller bronchi and vessels are effaced in the areas of scarring, or caseation which have almost completely replaced the alveolar structure throughout both lobes. *2*, Composite low-power photograph of the histologic preparations of the lung. The cavities noted in *1* are again seen in the upper lobe, and the virtually complete replacement of alveolar structure by scarring and caseation is apparent. The elongated structures with serrated linings are bronchi, chiefly of the second order. Many of these are dilated, and are embedded in areas of scarring. The more smoothly outlined spaces are vessels of the pulmonary system. Smaller branches of both the bronchi and the vessels are effaced in the scar tissue or caseous material. (Approximately ×1.5.) *3*, Photomicrograph of the wall of one of the bronchi showing dilatation and serration of the lining. Note that the mucosa is intact. There is moderate lymphocyte infiltration beneath the mucosa. At the left is shown a portion of the wide area of diffuse collagen deposition through which the bronchus courses. All evidences of bronchioles, alveolar ducts, and alveoli are effaced. Compare with Fig. 5, *4* which shows localized peribronchial scarring with preservation of adjacent alveolar structure. *4*, Photomicrograph of a normal bronchus of similar size to those in Figs. 4 and 8. Note the cartilagenous plate at the left. The epithelium is intact.

and the bronchial change seen was small in degree. On the other hand, the bronchographic pattern remained normal in areas where the parenchyma was not diseased but the lung collapsed from without, as in cases of pneumothorax (Cases 23 and 24), pleural effusion (Case 10), or thoracoplasty (Cases 17 and 21).

Bronchograms were also obtained in a number of patients who had pulmonary function studies (Cases 9, 13, 19, and 21) carried out preliminary to major chest surgery. It was hoped that some means of estimating function with the aid of bronchography might be worked out. To date insufficient material has been studied by both methods so that no conclusions can be

Fig. 9.—*1*, (*Case 10*) Posteroanterior roentgenogram of right lung showing linear shadows in upper field and density of residual pleural fluid at base. *2*, (*Case 10*) Posteroanterior bronchogram showing shortening, widening, and lack of arborization in upper lobe with no significant pattern changes in lower portion which is compressed by pleural fluid. *3*, (*Case 11*) Posteroanterior roentgenogram showing density at apex. Question as to whether this represents contracted segment or lobe. *4*, (*Case 11*) Bronchogram showing shadow representing entire right upper lobe contracted.

1. 2. 3.



4. 5.

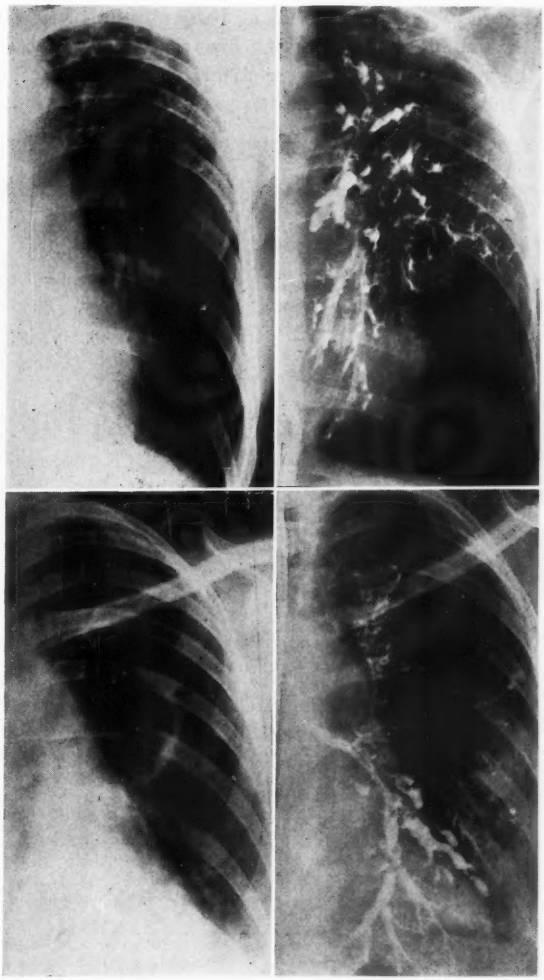
Fig. 10.—*1,* (*Case 12*) Posteroanterior roentgenogram showing extensive abnormal shadows in upper lung field. *2* and *3,* Posteroanterior and right lateral bronchogram showing characteristic short, wide bronchi in most of upper lobe and apex of lower lobe. *4,* (*Case 13*) Posteroanterior roentgenogram showing small annular shadow in upper lung field, left and mediastinal displacement. *5,* (*Case 13*) Bronchogram showing very poor filling throughout most of the left lung except at base despite every effort to improve this by addition of contrast medium. This latter only resulted in filling of trachea, spill to opposite lung, and ingestion into esophagus.

Case 13.—Pulmonary Function Study

Estimated normal vital capacity	3.6 L.
Determined vital capacity	1.9 L.
Determined resting minute ventilation	9.2 L.
Determined maximum minute ventilation	68.0 L.

The right diaphragm moves normally, but the left has only slight movement. There is good clouding on the right in expiration, poor on the left and the mediastinum shifts to the right slightly. By fluoroscopy it is estimated that the right lung is doing 75 per cent of the ventilation.

1. 2.



3. 4.

Fig. 11.—*1*, (*Case 14*) Posteroanterior roentgenogram showing few scattered abnormal shadows in upper lung field and retracted root shadow. *2*, (*Case 14*) Posteroanterior bronchogram showing marked changes through most of the lung and revealing associated bronchial defects including emphysema. *3*, (*Case 15*) Posteroanterior roentgenogram showing few linear shadows at apex and giant-sized annular shadow identified as being in the lower lobe on lateral film. *4*, (*Case 15*) Posteroanterior bronchogram showing characteristic abnormal pattern in upper lobe and marked changes through the lower lobe. Only the lingular segment shows a normal pattern.

drawn. It might be stated, however, that while bronchospirometry reveals the function of each lung it does not indicate how that function is distributed between the different lobes or segments. Bronchographic studies reveal which lobes or segments have altered bronchi and the study of injected lung specimens indicate that changes noted in the bronchi are associated with similar alterations in the pulmonary arterial system. This has therefore been interpreted to mean that that portion of the lung must be functionally impaired.

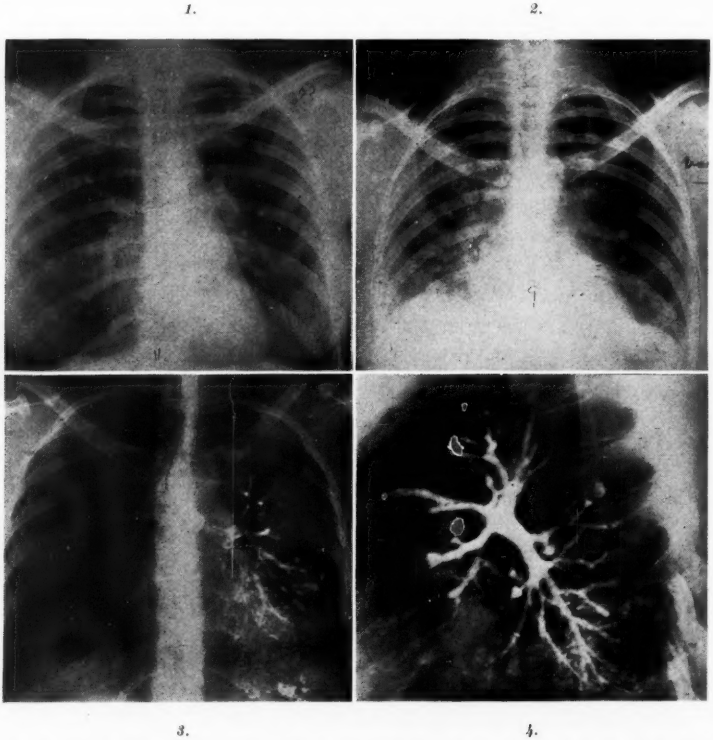


Fig. 12 (*Case 16*).—*1*, Posteroanterior roentgenogram taken at end of deep inspiration showing small annular shadow left upper lung field. *2*, Posteroanterior roentgenogram taken at end of forced expiration showing good clouding of right lung field and left lung field remaining dark indicating trapping of air. *3* and *4*, Posteroanterior and lateral bronchograms showing marked alteration of bronchial tree of entire upper lobe.

Bronchography, in conjunction with bronchospirometry, may be expected to show what portion of the lung is contributing the chief function present on a side.

From these studies it is believed that adequate bronchograms give much more information about the condition of the various parts of the lung than is generally apparent. It gives information which cannot be obtained by other methods of study, such as routine chest roentgenograms, including planigrams or bronchoscopy.

While routine chest roentgenograms give certain invaluable information regarding location and extent of disease, it is often impossible to tell from them exactly which segments are or are not diseased. When "clearing" occurs on routine chest roentgenograms, one cannot tell whether such resolution noticed by roentgenogram examination is accompanied by restitution of

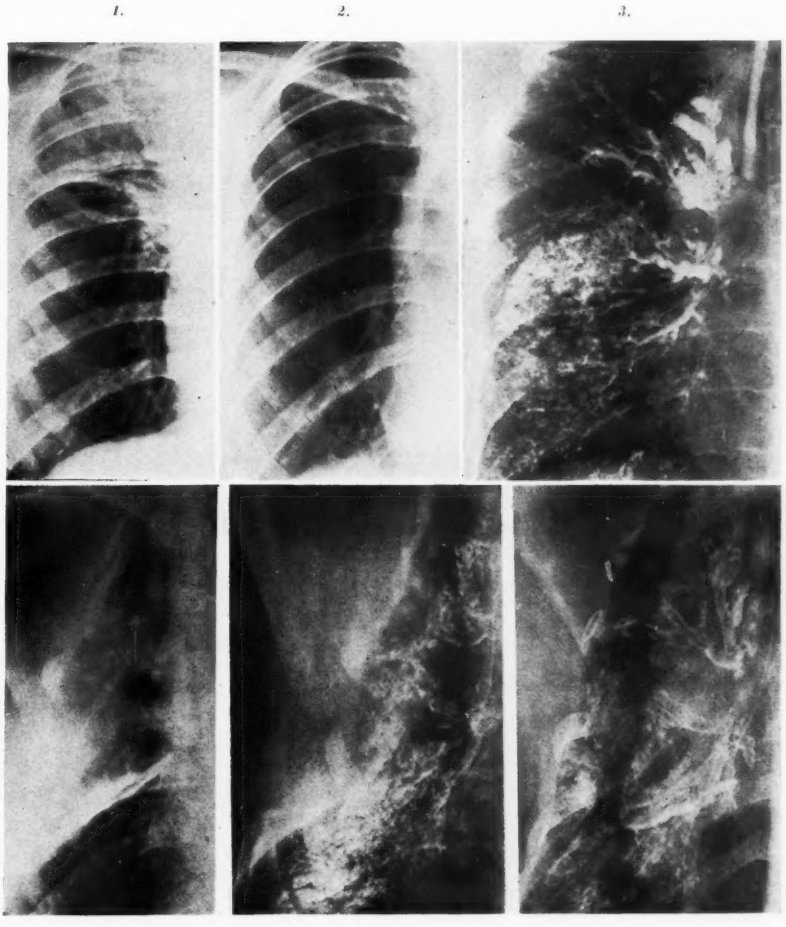
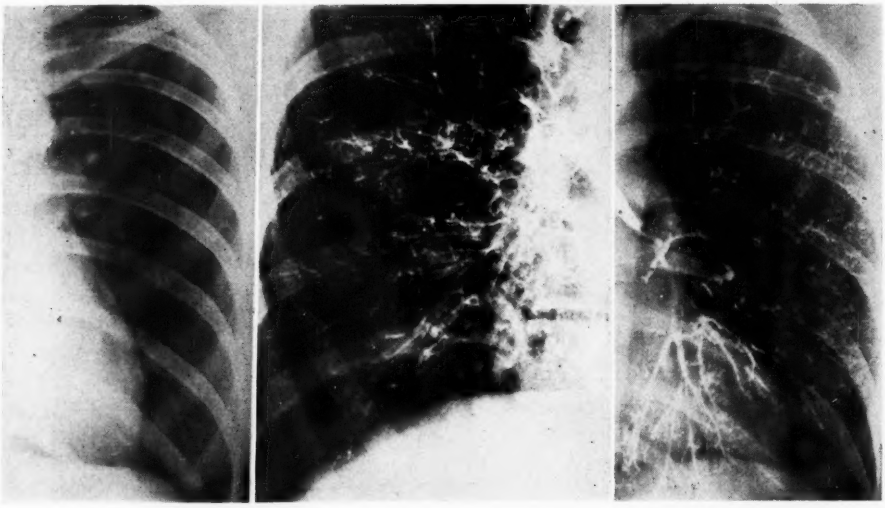


Fig. 13 (*Case 17*).—1, Posteroanterior roentgenogram showing dense shadows in right upper lung field at onset of illness in 1948. Patient treated with streptomycin and apparently recovered. *2*, Posteroanterior roentgenogram showing residual stringy shadows in right upper in 1950—sputum again positive. *3*, Posteroanterior bronchogram showing marked alteration of bronchial pattern in most of right upper lobe and apex of lower lobe. *4*, Posteroanterior roentgenogram following thoracoplasty. *5* and *6*, Posteroanterior and oblique bronchograms showing the persistence of changes noted before thoracoplasty.

The studies suggest that changes seen in the postthoracoplasty bronchograms may have been present before operation. Only bronchogram films taken before and after operation permit drawing any conclusions regarding the influence of thoracoplasty collapse on the bronchial tree.

normal anatomy. That is, one cannot tell to what extent the previously diseased lung has been damaged, even though the tissue contains air and is therefore radiolucent. In inexpandible lungs, as sometimes occurs after pneumothorax, the routine chest films give very little information regarding the condition of the collapsed lung, while bronchograms still distinguish diseased areas in such cases in the collapsed as in the uncollapsed lung (Cases 22, 23, and 25). Planigraphic studies of the chest are distinctly more valuable than regular chest films, but at open chest operation it is not uncommon to find more disease than seen in the films and at times areas of disease not seen.



1. *2.* *3.*

Fig. 14 (Case 18).—*1*, Posteroanterior roentgenogram showing few shadows in left upper lung field. The right side was normal. *2* and *3*, Posteroanterior bronchograms showing right side, especially of lower lobe region which has a normal pattern in contrast to thin narrow tree with fewer branches at left base. At operation the lower lobe was found studded with discrete tuberculous nodules along the course of the bronchi and the involved segments of lung were excised. (Compare with Case 8, Fig. 4.)

Bronchoscopy gives valuable information in certain cases. However, its value is limited to the larger bronchi. Bronchography, in contrast, reveals the changes beyond the visual field of the scope.

The question of possible harmful effects is frequently raised when iodized oil is suggested for studying cases of pulmonary tuberculosis. The material has been used here for years in patients with both positive and negative sputum. A careful study of postbronchographic films over long periods has failed to show any harmful effects attributable to the iodized oil. When the examination is made with care and minimum quantities of oil are used, practically all of this is removed when the patient utilizes postural drainage after the roentgen films are taken.

The extensively diseased or collapsed lobe or lung is much smaller in size, and bronchial filling requires only a small amount of material. If continued effort is made to instill larger quantities of oil than needed, flooding of other

areas of the lung will result. Retention of some of the oil will then be noted for weeks or months, and may make the roentgenographic follow-up troublesome in interpreting spreads of disease. However, a careful study of films taken before and shortly after bronchography largely solves this problem.

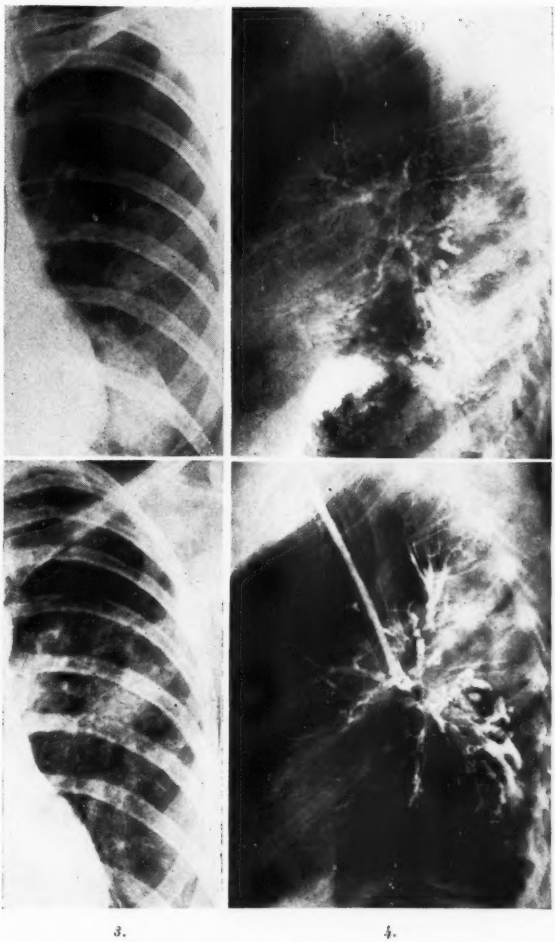


Fig. 15 (*Case 19*).—*1*, Posteroanterior roentgenogram showing abnormal density in lower lung region. *2*, Lateral bronchogram showing marked contraction of entire lower lobe with short, wide bronchi and few branches—the upper lobe shows some distension but a relatively normal bronchial pattern. *3*, Posteroanterior roentgenogram showing new shadows in mid lung field. *4*, Lateral bronchogram showing alteration of bronchial tree in region of new disease which previously showed normal pattern.

Case 19.—Pulmonary Function Study

Estimated normal vital capacity	3.25 L.
Determined vital capacity	3.15 L.
Determined resting minute ventilation	11 L.
Maximum minute ventilation	92 L.

By fluoroscopy the function on the right is normal, and the left lung functions fairly well except the lower lobe.

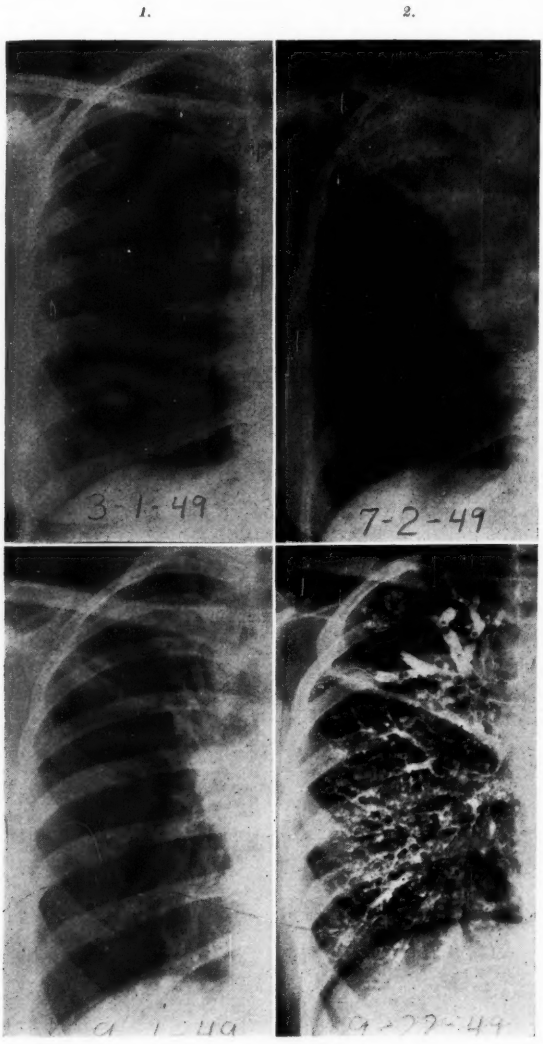
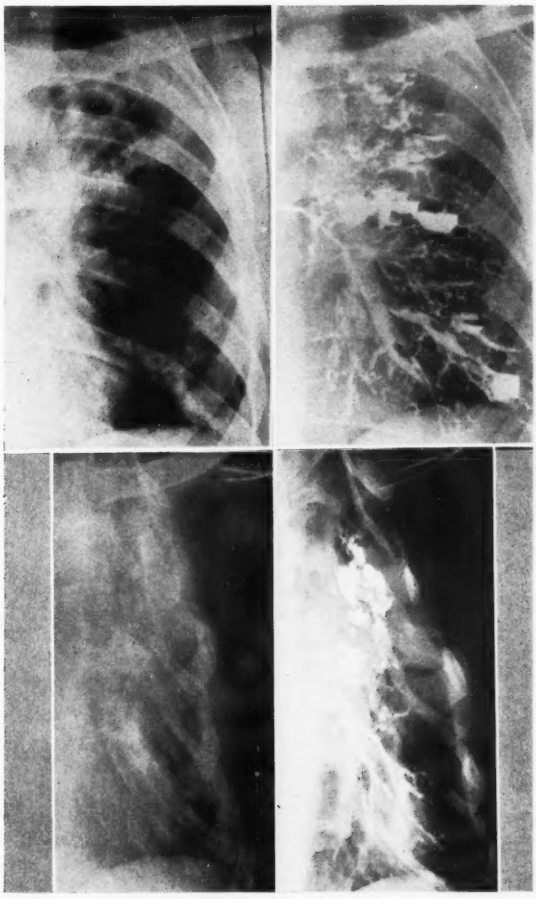


Fig. 16 (*Case 20*).—*1*, Posteroanterior roentgenogram, March 1, 1949, of patient with known minimal disease in extreme apex. *2*, Posteroanterior roentgenogram, July 2, 1949, after recent onset of tuberculous pneumonia showing dense wedge shaped shadow. *3*, Posteroanterior roentgenogram, Sept. 1, 1949, showing marked clearing of shadows with aeration of upper lung region and few linear strands. Film taken after two months treatment with streptomycin and PAS. *4*, Posteroanterior bronchogram, Sept. 22, 1949, showing marked changes in bronchial pattern of upper lobe. The rest of the tree appears normal.

1. 2.



3. 4.

Fig. 17 (*Case 21*).—*1*, Posteroanterior roentgenogram showing annular shadows in upper lung field and at base. *2*, Posteroanterior bronchogram showing shadows of cystic abnormalities as well as alteration of bronchi. *3*, Posteroanterior roentgenogram—postthoracoplasty. *4*, Posteroanterior bronchogram showing persistence of abnormal shadows of bronchial tree after thoracoplasty.

Case 21.—Pulmonary Function Study Before Thoracoplasty

Estimated normal vital capacity	4.8 L.
Determined vital capacity	3.05 L.
Determined resting minute ventilation	16 L.
Determined maximum minute ventilation	120 L.

The diaphragms move well on both sides and the clouding during expiration is good on the right, but fair to poor on the left. The mediastinum shifts to the right on expiration. The greatest part of ventilation is being done by the right lung as studied by fluoroscopy.

Ten Months Postthoracoplasty

Estimated normal vital capacity	5.25 L.
Determined vital capacity	4.9 L.
Determined resting minute ventilation	9 L.
Determined maximum minute ventilation	92 L.

The diaphragm on the right moves well and there is good clouding on the right except at the apex. The diaphragm on the left moves poorly and the mediastinum shifts to the right on expiration. There is almost no ventilation on the left as studied by fluoroscopy.

After a few trial cases one can readily develop the necessary skill to be satisfied that the absence or poor filling of the bronchi is not due to technical fault. The abnormal bronchographic pattern in extensive parenchymal involvement so well depicted in Cases 12, 17, and 20 and in discrete dissemination in Cases 16, 18, and 19 need not be confused with incomplete filling of normal bronchi which would show normal caliber and some normal arboriza-

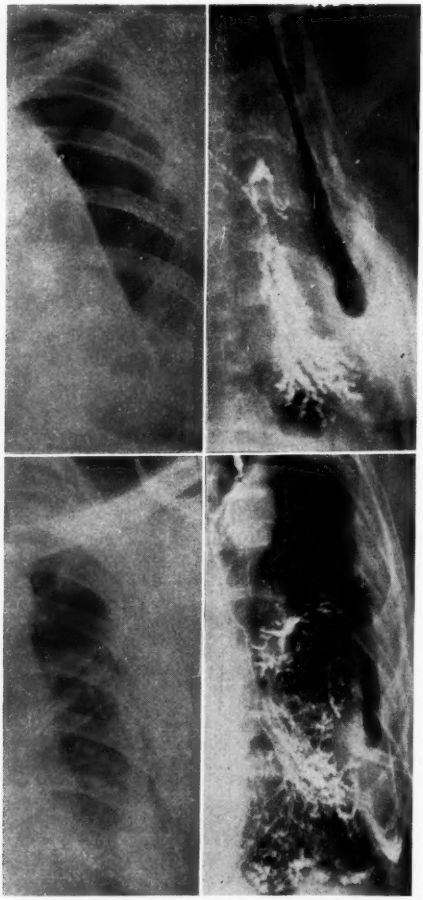


Fig. 18.—*1*, (*Case 22*) Posteroanterior roentgenogram showing totally collapsed lung proved to be inexpandible. *2*, (*Case 22*) Posteroanterior roentgenogram postthoracoplasty showing residual space and bronchogram showing poor pattern generally. Two attempts at reexpansion by decortication failed. *3*, (*Case 23*) Posteroanterior roentgenogram showing residual pleural space at base in case of oleothorax and a known bronchopleural fistula and empyema. *4*, (*Case 23*) Posteroanterior bronchogram showing normal pattern in lower lobe and shadow of catheter draining pleural space. Decortication and repair of fistula resulted in obliteration of pleural space by expansion.

tion. Patients with actively progressive disease or excessive secretions should not be chosen for this examination because of risk of spread of disease or retention of secretions when the cough reflex is temporarily blocked by topical anesthesia (0.5 per cent pontocaine solution).

It is helpful to have bronchoscoped the patient sometime before to ascertain the condition of the bronchial tree, noting any obstructive lesions and the disposition of the orifices to the lobes. This serves as a guide in positioning

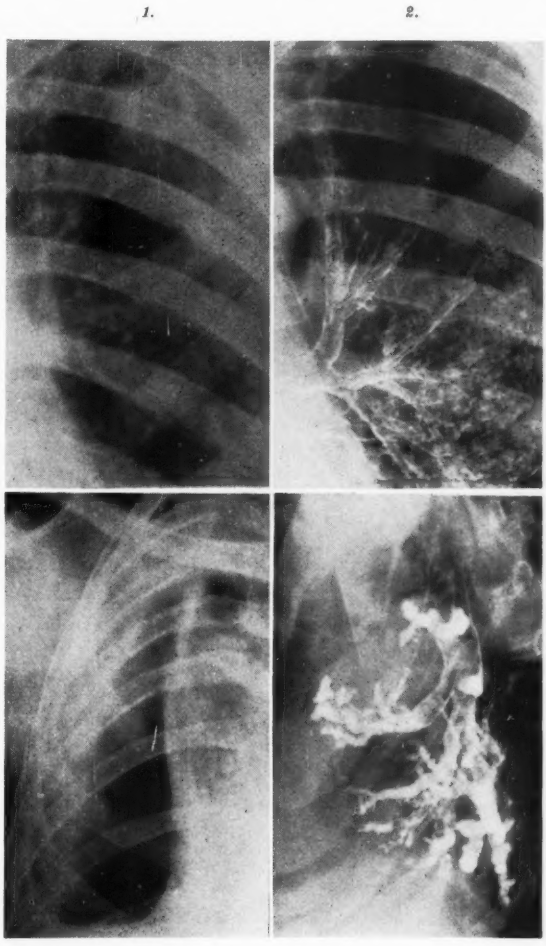


Fig. 19.—*1*, (*Case 24*) Posteroanterior roentgenogram showing annular shadow in upper lung field. *2*, (*Case 24*) Posteroanterior bronchogram showing abnormal bronchi in the involved part of lung only and no change elsewhere despite presence of pneumothorax. *3*, (*Case 25*) Posteroanterior roentgenogram of unexpandible lung in presence of pyopneumothorax. *4*, (*Case 25*) Oblique bronchogram showing marked alteration of bronchi to all lobes, indicating that reexpansion may not be anticipated with decortication.

the patient during the bronchographic examination. The combination of bronchoscopy and instillation of iodized oil is not advised, as it has not met with the success of doing the latter as a separate procedure at another time. An exception to this is the patient with a high degree of stenosis of a main

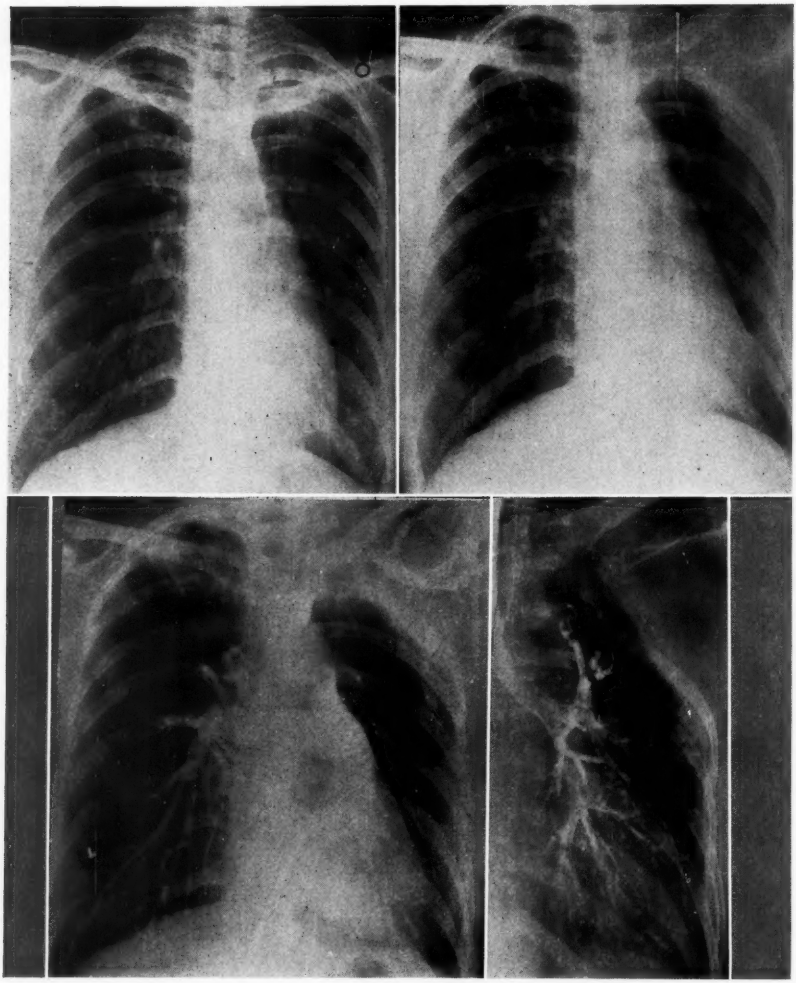


Fig. 20 (*Case 26*).—*1*, Posteroanterior roentgenogram showing small annular shadow left upper. *2*, Posteroanterior roentgenogram following first stage thoracoplasty. *3*, Posteroanterior angiocardiogram showing decrease in pulmonary arterial pattern in left lung and right upper which is site of old disease. *4*, Posteroanterior bronchogram of left showing decrease in arborization left upper and superior segment of lower. At operation disease involved much of apical-posterior segment of upper and superior segment of lower which were excised.

bronchus. Here the bronchoscopy is done on the roentgenogram table and, with the roentgenologist present, a watery contrast medium is instilled under fluoroscopic control. "Spot films" are taken followed by the immediate aspiration of the material and secretions. The use of nonoily solutions for

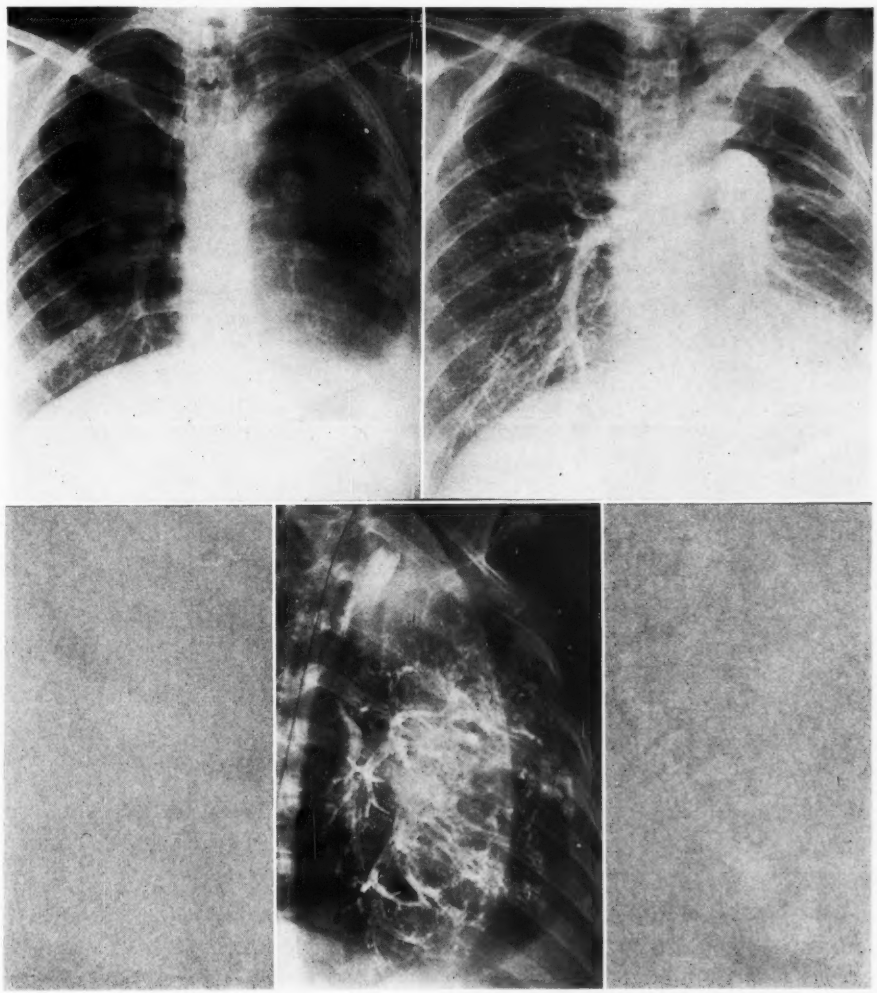


Fig. 21 (*Case 27*).—*1*, Posteroanterior roentgenogram in case of reexpanded (?) left pneumothorax showing mediastinal displacement cortical peel and linear density at site of previous cavity. *2*, Posteroanterior angiocardiogram showing fairly good pattern of left pulmonary artery except at linear density. Pulmonary arterial pattern on right is normal. *3*, Oblique bronchogram showing good pattern in all lobes except in same region as linear density where pulmonary arterial pattern was also decreased.

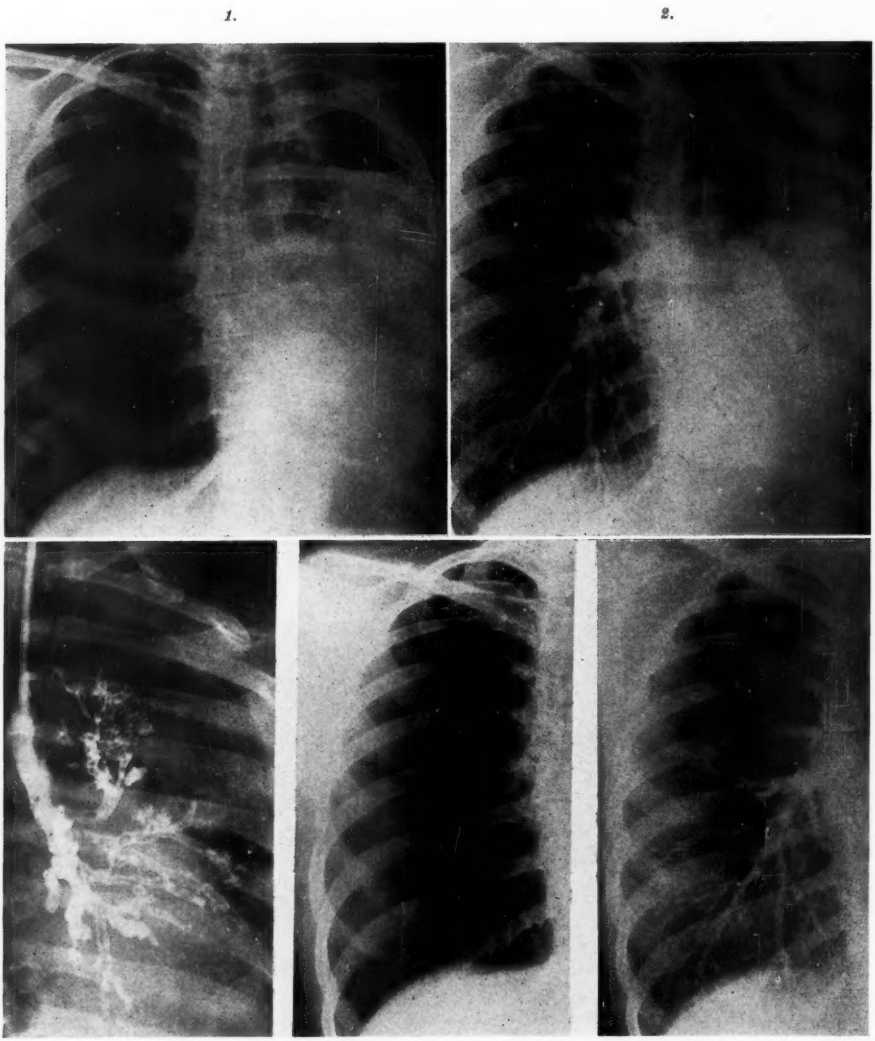


Fig. 22 (*Case 28*).—*1*, Posteroanterior roentgenogram showing unexpandible left lung with pyopneumothorax. *2*, Posteroanterior angiocardiogram showing absence of pattern of left pulmonary artery. *3*, Posteroanterior bronchogram left showing very poor bronchial pattern in all segments. *4*, Posteroanterior roentgenogram of right showing nodular densities in left upper lung field. *5*, Posteroanterior angiogram right side showing poor pulmonary arterial pattern in same area in contrast to normal pattern in middle and lower lobes.

routine bronchograms is not recommended, as it has not shown sufficiently satisfactory detail in the roentgenograms to be generally employed and is only used in the above situation. It is felt that bronchography gives such important information that certain disadvantages are outweighed. The examination can be a distinct aid in planning surgical treatment for pulmonary tuberculosis. The following are cited as illustrative examples of how bronchography can be helpful.

1. When resection of lung tissue is contemplated bronchograms aid in accurate localization of disease and thus help in deciding exactly which lobes or/and segments of lobes may need to be removed.

2. When thoracoplasty is considered it aids in helping to decide how extensive a procedure should be performed. Bronchograms frequently show evidence of marked structural changes, undoubtedly associated with damaged lung tissue, in areas where "clearing" has taken place as interpreted by routine chest roentgenograms. It is believed that such cases might fare better under permanent collapse (exemplified by Case 17). Perhaps prethoracoplasty bronchograms might also give a clue as to why some thoracoplasties are successful while others are not.

3. When decortication is considered to allow a "non-reexpansible" lung under pneumothorax to expand once more, bronchography gives the best information available as to the extent of structural changes present in the collapsed lung and thus aids in deciding whether the lung is healthy enough to make decortication advisable. (Exemplified by Cases 22, 23, and 25).

A PRELIMINARY NOTE ON PULMONARY ANGIOGRAPHY

Recently, with the aid of Dr. Charles T. Dotter, Radiologist at the New York Hospital, New York City, the technique of angiocardiography has been added. Dr. Berkeley Zinn, Supervising Radiologist at the Ray Brook State Tuberculosis Hospital, Ray Brook, N. Y., undertook a period of training under Dr. Dotter and returned here to add this method of study to the radiographic armamentarium. A study is under way to compare the pulmonary angiograms with bronchograms. Where possible injection study of suitable specimens as outlined in the early part of this paper will then be made before pathologic examinations are carried out and all material then correlated. Thus far, in a small series of cases, the anticipated correlation on the basis of injection studies of bronchi and pulmonary arteries has been observed. In Case 26 abnormal shadows of pulmonary tuberculosis were seen in both apices. A thoracoplasty was planned to collapse the small cavity in the summit of the left lung where an annular shadow was noted. After a one-stage, three-rib thoracoplasty was done an angiogram and bronchogram were performed at different times which indicated in both more structural alteration of the lung than suspected. On this basis pulmonary resection was chosen as the procedure of choice, and at operation the involved parts of the left upper and superior segment of the lower lobes were removed. Cases 27 and 28 were studied preliminary to consideration of decortication. Because of such good bronchial and pulmonary arterial

patterns in the former decortication with limited resection of the involved portion of the lung was chosen in Case 27. In Case 28, because of widespread change in the bronchogram and very poor filling of the left pulmonary artery, pleuropneumonectomy was considered the procedure of choice.

SUMMARY

Tuberculous lung specimens, which were removed at operation or autopsy, were studied by injecting the bronchi and pulmonary arteries with radiopaque material. Pathologic studies of these lungs were made showing the extent of disease present. This study served as a basis for the interpretation of bronchograms in the living. It is believed that bronchograms give more information than is generally apparent as to the exact location and extent of disease present. It may also suggest the amount of functionally available lung tissue still present. Recently, angiocardiographic studies have been added in addition to bronchograms, and appear to correlate well with the injected lung studies that both systems are implicated in the structural alterations of the lung by chronic pulmonary tuberculosis. This information has been used in planning such types of surgical treatment of pulmonary tuberculosis as resection, thoracoplasty, and decortication. Numerous illustrations are included.

A note of thanks is made to the many physicians and surgeons in the Saranac Lake area for their excellent cooperation in permitting the use of surgical and postmortem specimens for study. Thanks are also extended to the resident staff members and technicians who cooperated in this work.

The radiographic illustrations were prepared by Anna M. Sanlow, Senior X-ray Technician, Ray Brook State Tuberculosis Hospital, Ray Brook, N. Y.

The pathologic illustrations were prepared by Edward Gockeler, Photographer, Saranac Laboratory, Saranac Lake, N. Y.

EFFECT OF POSITION AND ARTIFICAL VENTILATION ON THE EXCRETION OF CARBON DIOXIDE DURING THORACIC SURGERY

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BOSTON, MASS.

OUR finding[3] that a dangerously high tension of carbon dioxide is common in the blood during open thoracic surgery has been widely confirmed.[6-9] It is now generally agreed that the tension of carbon dioxide will rise seriously during surgery with the pleura open unless active measures are taken to prevent it. We are not all agreed, however, as to the reason for this accumulation of carbon dioxide, nor are we all agreed as to the best method to get rid of the excess carbon dioxide. The purpose of this study was to try to answer the questions of why the carbon dioxide tension rises and of how to prevent this rise.

Elimination of carbon dioxide has been studied during thoracic surgery in the prone position used by Overholt and in the supine position, as well as in the lateral position with and without artificial assistance of ventilation. We showed in our original paper[3] that the pneumonectomy cases present the greatest problem. Therefore we have focused our principal attention on this group. A comparison of the data obtained when the patients are in the several positions demonstrates that position of the patient plays the major role in the development of or avoidance of a respiratory acidosis, in the absence of artificial respiration. Several other factors add to the patient's respiratory embarrassment: the existing pulmonary disease, the anesthesia, the open pleura, the crippling imposed during surgery; but these are, of course, present in all positions and cancel out in the comparisons.

Comparison of the work of the several groups now interested in this field permits some tentative conclusions as to the relative effectiveness of the two principal methods of artificial ventilation of the lungs, the hand and the machine.

We have shown already that it is easy to maintain full oxygenation in nearly all cases when patients are breathing the 96 volumes per cent oxygen possible with ether. Carbon dioxide excretion is the great problem; it is the subject of this paper.

METHODS

Conduct of Anesthesia.—Premedication in most instances consisted of pentobarbital sodium 100 or 200 mg., morphine 10 or 15 mg., and atropine 0.6 mg.

From the Anesthesia Laboratory of the Harvard Medical School at the Massachusetts General Hospital.

This work was supported in part by an Institutional Grant from the American Cancer Society and by a gift from Mr. Edward Mallinckrodt. For this help we express our sincere thanks.

Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery at Atlantic City, N. J., April 16-18, 1951.

The subjects to be operated on in the prone position received the higher dosages mentioned, and this can be assumed to account for the somewhat higher preanesthetic carbon dioxide tensions in this group.[5]

Patients in the lateral group* were induced with 80 volumes per cent nitrous oxide and 20 volumes per cent oxygen followed by endotracheal ether-oxygen maintenance in a closed system with "circle" soda lime filter. Patients in the prone (Overholt) position were intubated under topical anesthesia and placed in the prone position. General anesthesia was then induced with cyclopropane and oxygen. Anesthesia was maintained with ether and oxygen. The respiration was assisted actively by the hand-bag-squeeze method in all patients in the lateral position. An important limiting factor in the extent to which assistance can be given to the respiration is how much mechanical disturbance the surgeon can tolerate during his work within the chest. In the prone and supine patients ventilation was not assisted.

The Biochemical Determinations and Calculations were carried out as described in our earlier papers.[1-3]

RESULTS AND DISCUSSION

The data are presented in Tables I to V, and are summarized in Table VI. It is evident that elimination of carbon dioxide in the prone position *without* assistance of respiration was as good in the case of lobectomies and better in the case of pneumonectomies than in the lateral position with bag squeezing or machine (see Table VIII) assistance to respiration. Not enough patients have yet been studied in the supine position for adequate evaluation; but it appears that patients on their backs as well as on their bellies (Overholt) are in a more favorable position for the natural excretion (unassisted ventilation) than they are in the lateral position.

Thus position is a prime factor in the rise in carbon dioxide during thoracic surgery. We are unable to agree with Taylor and Roos[9] that this rise is due to the action of ether on the respiratory center. In the first place, ether is a respiratory stimulant, not a depressant as far as minute volume of respiration goes, at the levels of anesthesia needed for thoracic surgery. Second, no such carbon dioxide accumulation need occur in non-thoracic patients under ether. We[2] showed in twenty patients undergoing surgery in other parts of their bodies but during ether anesthesia and in the supine position that there was no accumulation of carbon dioxide. This has more recently been confirmed in poor risk patients with advanced cardiac, respiratory, and metabolic disease.[4] Actually, these patients[2] blew off carbon dioxide and the pCO_2 was a little below normal at the end of the operation, illustrating the stimulant effect of ether on the respiration.

Nor can we agree with Gibbon and his associates[6] that the rise in carbon dioxide tension is "largely a result of the open thorax, and not the lateral position upon the operating table." In the cases presented here the pleura was open in the prone and in the supine cases, yet without any such tendency for the pCO_2

*See our previous paper[3] for a full description of placement of these patients.

TABLE I. LOBECTOMY, LATERAL POSITION—ASSISTED RESPIRATION

SEX	AGE (YR.)	OPERATION	ANESTHESIA TIME (MIN.)	PLEURA OPEN (MIN.)	PH	CO_2 TENSION (MM. HG)	SERUM CO_2 (MM./L.)	$[HCO_3^-]$ (MM./L.)	HEMOGLOBIN (GM. %)	β	CORRECTION	$[HCO_3^-]$ CORRECTED TO PH 7.40	CHANGE IN FIXED ACID
M	48	Rt. U. Lobect.	230	160	7.43*	39	26.4	25.0		(28)	+0.8	25.8	
					7.37*	34	20.6	19.8		(28)	−0.8	19.0	+1.8
M	51	Lt. L. Lobect.	145	95	7.43	41	28.3	27.0	14.3	28.3	+0.8	27.8	
					7.35	46	27.0	25.2	10.0	22.6	−1.1	24.1	+3.7
F	43	Rt. M. Lobect	245	180	7.40	43	25.5	24.0	12.2	25.6	0	24.0	
					7.40	37	23.9	22.7	9.4	21.7	0	22.7	+1.3
F	51	Rt. L. Lobect.	183	95	7.40	44	28.3	26.7	10.8	23.8	0	26.7	
					7.38	45	27.6	26.0	10.9	23.8	−0.5	25.5	+1.2
M	25	Rt. L. Lobect.	180	120	7.42	44	29.2	27.5	12.4	25.9	+0.5	28.0	
					7.38	47	28.5	27.0	12.2	25.6	−0.5	26.5	+1.5
M	66	Rt. M. Lobect.	134	75	7.37	47	28.1	26.4	14.3	28.3	−0.8	25.6	
					7.32	49	26.2	24.8	14.6	29.1	−2.3	22.5	+3.1
F	20	Lt. L. Lobect.	300	225	7.35	49	28.5	26.8	13.4	27.3	−1.4	25.4	
					7.30	56	29.0	27.1	11.8	25.1	−2.5	24.6	+0.8
M	67	Rt. U. Lobect.	115	60	7.45	39	28.0	26.6	13.1	26.9	+1.4	28.0	
					7.43	40	27.0	25.8	12.9	26.5	+0.8	26.6	+1.4
M	62	Rt. U. Lobect.	255	170	7.41	50	32.6	30.9	14.0	28.2	+0.3	31.2	
					7.20	87	36.4	33.8	13.8	27.8	−5.6	28.2	+3.0
F	45	Rt. M. and L. Lobect.	217	155	7.45	40	28.9	27.5	14.1	28.4	+1.4	28.9	
					7.40	35	22.0	21.0	14.1	28.4	0	21.0	+7.9
Average	49		200	134	7.41	44	28.4	26.8	13.9	27.0	+0.3	27.1	
					7.35	48	26.8	25.3	12.2	27.0	−1.3	24.1	+3.0

*Upper line, preanesthetic; lower line, near end of anesthesia.

TABLE II. LOBECTOMY, PRONE (OVERHOLT) POSITION—NOT ASSISTED

SEX	AGE (YR.)	OPERATION	ANESTHESIA TIME (MIN.)	PLEURA OPEN (MIN.)	PH	CO_2 TENSION (MM. HG)	SERUM CO_2 (MM./L.)	$[HCO_3^-]$ (MM./L.)	HEMOGLOBIN (GM. %)	β	CORRECTION	$[HCO_3^-]$ CORRECTED TO PH 7.40	CHANGE IN FIXED ACID
F	53	Rt. M. Lobect.	95	50	7.30*	48	24.7	23.0	14.4	28.7	−2.9	20.1	
					7.30*	52	26.8	25.0	14.9	29.3	−2.9	22.1	−2.0
M	61	Rt. U. Lobect.	150	70	7.35	50	28.6	27.0	15.1	29.7	−1.5	25.5	
					7.30	49	25.5	24.0	14.4	28.7	−2.9	21.1	+4.4
F	35	Lt. U. Lobect.	155	85	7.28	54	27.0	25.2	11.7	24.9	−3.0	22.2	
					7.23	56	24.6	22.9	12.1	25.4	−4.3	18.6	+3.6
F	36	Rt. L. Lobect.	130	70	7.45	28	20.0	19.3	12.2	25.6	+1.3	20.6	
					7.40	37	23.5	22.2	11.6	24.8	0	22.2	−1.6
M	55	Rt. U. Lobect.	190	130	7.37	48	29.1	27.3	14.3	28.3	−0.8	26.5	
					7.33	46	25.2	23.6	13.7	27.6	−1.9	21.7	+4.8
F	54	Rt. M. Lobect.	130	80	7.33	41	22.3	21.1	13.8	27.9	−2.0	19.1	
					7.30	45	23.4	22.0	11.5	24.3	−2.4	19.6	−0.5
M	73	Rt. U. Lobect.	145	100	7.41	43	28.5	27.0	14.4	28.7	+0.3	27.3	
					7.33	47	25.7	24.0	10.0	22.6	−1.6	22.4	+4.9
F	50	Rt. M. Lobect.	80	40	7.38	45	27.8	26.0	12.2	25.6	−0.5	25.5	
					7.18	72	28.8	26.2	6.8	18.0	−4.0	22.2	+3.3
M	43	Rt. L. Seg. Lobect.	154	80	7.30	48	24.5	23.0	14.9	29.3	−2.9	20.1	
					7.27	51	24.5	23.0	14.6	29.0	−3.8	19.2	+0.9
M	50	Seg. Lobect.	130	80	7.30	52	26.9	25.0	14.9	29.3	−2.9	22.1	
					7.33	45	24.6	23.1	14.8	29.3	−2.1	21.0	+1.1
M	59	Rt. M. Lobect.	145	65	7.33	48	26.3	24.8	14.2	28.3	−1.3	23.5	
					7.27	47	22.5	21.0	14.1	28.4	−3.7	17.3	+6.2
M	58	Rt. U. Lobect.	205	160	7.35	52	29.8	27.9	13.0	26.8	−1.3	26.6	
					7.25	66	30.7	28.4	12.9	26.7	−4.0	24.4	+2.2
			142	84	7.35	46	26.3	24.7	13.8	27.8	−1.5	23.2	
Average	52				7.29	51	25.5	23.8	12.6	26.2	−2.8	21.0	+2.2

*Upper line, preanesthetic; lower line, near end of anesthesia.

TABLE III. PNEUMONECTOMY, LATERAL POSITION—ASSISTED RESPIRATION

SEX	AGE (YR.)	OPERATION	ANESTHESIA TIME (MIN.)	PLEURA OPEN (MIN.)	PH	CO_2 TENSION (MM. HG)	SERUM CO_2 (MM./L.)	$[HCO_3^-]$ (MM./L.)	HEMOGLOBIN (GM. %)	β	CORRECTION	$[HCO_3^-]$ CORRECTED TO PH 7.40	CHANGE IN FIXED ACID
M	70	Lt. Pneumonect.	215	165	7.45*	39	28.3	26.7	12.2	25.6	+1.3	28.0	
					7.25*	65	30.2	28.0	12.2	25.6	−3.8	24.2	+4.8
M	63	Rt. Pneumonect.	220	155	7.40	37	24.0	22.8	11.8	25.1	--	22.8	
					7.40	37	23.3	22.2	12.2	25.6	--	22.2	+0.6
M	56	Rt. Pneumonect.	210	130	7.40	40	25.6	24.1	12.8	26.4	--	24.1	
					7.25	63	29.1	27.0	13.1	26.9	−4.0	23.0	+1.1
M	63	Rt. Pneumonect.	160	115	7.27	56	27.2	25.2	14.6	29.1	−3.8	21.4	
					7.27	59	28.3	26.4		(28)	−3.6	22.8	−1.4
M	16	Lt. Pneumonect.	118	60	7.37	46	27.7	26.0	15.2	30.0	−0.9	25.1	
					7.30	45	22.8	21.2	15.0	29.5	−3.0	18.2	+6.9
M	56	Lt. Pneumonect.	275	205	7.37	47	28.3	26.8	12.2	25.6	−0.8	26.0	
					7.22	57	24.5	22.8	13.6	27.5	−5.0	17.8	+8.2
M	57	Rt. Pneumonect.	240	165	7.42	36	24.1	23.0	12.8	26.4	+0.5	23.5	
					7.18	60	23.6	21.8	13.0	26.7	−5.9	15.9	+7.6
M	54	Lt. Pneumonect.	180	130									
					7.20	74	31.0	28.8	14.3	28.6	−5.7	23.1	
Average	54		202	142	7.38	43	26.5	24.9	13.1	26.9	−0.5	24.4	
					7.25	58	26.6	24.8	13.3	27.2	−3.9	20.9	+3.9

*Upper line, preanesthetic; lower line, near end of anesthesia.

TABLE IV. PNEUMONECTOMY, PRONE (OVERHOLT) POSITION—NOT ASSISTED

SEX	AGE (YR.)	OPERATION	ANESTHESIA TIME (MIN.)	PLEURA OPEN (MIN.)	PH	CO_2 TENSION (MM. HG)	SERUM CO_2 (MM./L.)	$[HCO_3^-]$ (MM./L.)	HEMOGLOBIN (GM. %)	β	CORRECTION	$[HCO_3^-]$ CORRECTED TO PH 7.40	CHANGE IN FIXED ACID
F	62	Rt. Pneumonect.	150	100	7.33*	46	25.2	23.6		(28)	−2.0	21.6	
					7.27*	61	29.5	27.5		(28)	−3.6	23.9	−2.3
F	34	Lt. Pneumonect.	120	70	7.35	41	23.6	22.1	12.8	26.4	−1.3	20.8	
					7.30	45	22.5	21.1	12.3	25.8	−2.6	18.5	+2.3
F	29	Lt. Pneumonect.	115	70	7.37	40	23.8	22.3		(28)	−0.8	21.5	
					7.30	39	20.3	19.0		(28)	−2.8	16.2	+5.3
F	29	Lt. Pneumonect.	175	115	7.40	37	23.5	22.2	11.4	24.5	0	22.2	
					7.35	48	27.4	25.8	9.9	22.4	−1.1	24.7	−2.5
M	57	Lt. Pneumonect.	115	65									
					7.32	52	27.7	25.9	14.2	28.5	−2.3	23.6	
F	40	Lt. Pneumonect.	105	65	7.33	40	22.1	20.9	13.7	27.8	−1.9	19.0	
					7.28	46	22.6	21.1	13.6	27.5	−3.3	17.8	+1.2
M	34	Lt. Pneumonect.	145	75	7.36	41	24.1	22.8	11.7	24.9	−1.0	21.8	
					7.36	40	23.1	21.9	15.2	30.0	−1.2	20.7	+1.1
F	32	Lt. Pneumonect.	145	70	7.38	42	25.6	24.1	13.3	27.3	−0.5	23.6	
					7.33	44	24.3	23.0	12.2	25.6	−1.8	20.2	+3.4
F	28	Lt. Pneumonect.	60	45	7.35	44	25.2	23.9	11.3	24.3	−1.2	22.7	
					7.30	61	31.6	29.5	11.7	24.9	−2.5	27.0	−4.3
M	62	Rt. Pneumonect.	90	55	7.41	36	23.6	22.3	13.3	27.2	+0.3	22.6	
					7.39	47	24.4	22.9	13.8	27.9	−2.8	20.1	+2.5
M	52	Lt. Pneumonect.	160	120	7.40	37	23.9	22.5	12.5	26.1		22.5	
					7.38	38	23.0	21.7	12.8	26.5	−0.5	21.2	+1.3
F	38	Lt. Pneumonect.	105	45	7.34	48	27.0	25.4	12.0	25.4	−1.5	23.9	
					7.29	48	24.6	23.0	11.9	25.3	−2.8	20.2	+3.7
Average	41		124	75	7.37	41	24.3	22.8	12.4	26.0	−0.9	22.0	
					7.32	47	25.1	23.5	12.8	26.4	−2.3	21.2	+1.1

*Upper line, preanesthetic; lower line, near end of anesthesia.

TABLE V. MISCELLANEOUS PROCEDURES, OPEN CHEST, SUPINE—NOT ASSISTED

SEX	AGE (YR.)	OPERATION	ANESTHESIA TIME (MIN.)	PLEURA OPEN (MIN.)	PH	CO_2 TENSION (MM. HG)	SERUM CO_2 (MM./L.)	$[HCO_3^-]$ (MM./L.)	HEMOGLOBIN (GM. %)	β	CORRECTION	$[HCO_3^-]$ CORRECTED TO PH 7.40	CHANGE IN FIXED ACID
F	60	Lt. Pneumonect.	225	145	7.35*	48	27.3	24.8	14.9	29.3	−1.5	23.3	
					7.35*	42	24.2	22.8	14.8	29.1	−1.5	21.3	+2.0
F	65	Exploration (Inoperable)	(130)	(60)	7.38	42	25.4	24.0	12.9	26.7	−0.5	23.5	
					7.37	43	25.8	24.4	12.9	26.7	−0.8	23.4	+0.1
F	38	Rt. M. Lobect.	120	65	7.41	40	26.6	25.1	14.5	28.9	+0.3	26.9	
					7.27	(57)	(27.8)	(25.8)	13.9	28.1	−3.7	(24.1)	(+2.8)
M	54	Rt. M. Lobect.	140	70	7.39	43	27.3	25.6	16.1	31.2	−0.3	25.3	
					7.31	53	28.3	26.4	15.0	29.6	−2.7	23.7	+1.6
F	52	Lingulectomy	110	60	7.40	46	29.8	28.0	15.2	29.9	0	28.0	
					7.35	45	25.7	24.2	14.0	28.6	−1.4	22.8	+5.2
F	32	Exc. Pericard. Cyst	52	22									
					7.29	45	22.7	21.3	12.8	26.7	−2.9	18.4	
M	63	Lt. Pneumonect.	170	120	7.41	44	29.0	27.3	12.6	26.2	+0.3	27.6	
					7.45	36	25.5	24.3	12.0	25.4	+1.3	25.6	+2.0
Average	52		135	77	7.39	44	27.6	25.2	14.4	28.7	−0.3	25.8	
					7.34	46	25.7	24.2	13.6	27.7	−1.7	22.8	+2.3

*Upper line, preanesthetic; lower line, near end of anesthesia.

TABLE VI. EFFECT OF POSITION ON CARBON DIOXIDE TENSION

	PATIENTS (NO.)	AGE (YR.)	PLEURA OPEN (MIN.)	pH	SERUM CO_2 (MM./L.)	CO_2 TENSION (MM. HG)
Lobectomy						
Lateral position*	8	34	96	7.51	25.9	33
Not assisted				7.22	26.2	67
Lobectomy						
Lateral position	10	49	134	7.41	28.4	44
Assisted				7.35	26.8	48
Lobectomy						
Prone (Overholt)	12	52	92	7.35	26.6	46
Not assisted				7.29	25.5	51
Pneumonectomy						
Lateral position*	5	38	122	7.48	26.6	35
Not assisted				7.09	26.9	98
Pneumonectomy						
Lateral position	8	54	142	7.38	26.5	43
Assisted				7.26	26.6	58
Pneumonectomy						
Prone (Overholt)	12	41	75	7.37	24.3	41
Not assisted				7.32	25.1	47
Miscellaneous, Open Thorax						
Supine	7	52	77	7.39	27.6	44
Not assisted				7.34	25.7	46

*Beecher, H. K., and Murphy, A. J.[1]

TABLE VII. PNEUMONECTOMIES, PRONE, NOT ASSISTED (CHANGES OBSERVED DURING INDUCTION, OPENING OF PLEURA, ETC.)

PATIENT	ANESTHESIA TIME (MIN.)	PLEURA OPEN (MIN.)	pH	CO_2 TENSION (MM. HG)	SERUM CO_2 (MM./L.)	
H. B.	0	---	7.40	37	23.9	Preanesthetic
	20	---	7.30	41	21.3	Prone, no surgery
	(60)	(20)	7.32	45	24.5	Chest open
	160	120	7.38	38	23.0	Left lung out, chest open
L. D.	0	---	7.34	48	27.0	Preanesthetic
	30	---	7.28	53	26.4	Prone, no surgery
	75	15	7.27	49	23.9	Chest open
	105	45	7.29	48	24.6	Left lung out, chest open
	160	100	7.28	49	24.2	Chest closed

to rise. The only difference is the interference with the respiration produced by position.*

With the effect of position on the accumulation of carbon dioxide clearly established, we can then turn to a consideration of the best way to prevent the accumulation of it. Gibbon and his associates reported on thirteen patients undergoing pneumonectomy.[6] Notwithstanding artificial ventilation by machine four of the thirteen more than doubled their carbon dioxide tension; they showed a very high carbon dioxide tension, that is, above 90 mm. Hg at the end of operation. The levels in four patients out of thirteen are very high indeed and presumably dangerous. Their average carbon dioxide tension was 74 mm. Hg. Using hand assistance to respiration (bag squeezing) we had only one patient

*Serial determinations obtained during two of the prone pneumonectomies are presented in Table VII. These were particularly poor risk patients, and at no time while the pleura was open did either have a carbon dioxide tension above 50 mm. Hg, the accepted upper limit of normal. There was no change in carbon dioxide tension in samples drawn just prior to opening the pleura and just after opening the pleura.

this high and no other above sixty-five. Our average value for carbon dioxide tension in the assisted pneumonectomy group was 58 mm. Hg (see Table VIII). None of Overholt's pneumonectomies carried out in the prone position showed as much accumulation of carbon dioxide, *without any assistance to respiration.* We are obliged to conclude, on the evidence to date, that assistance to the respiration

TABLE VIII. COMPARISON OF MANUAL WITH MACHINE ASSISTANCE OF RESPIRATION (PNEUMONECTOMIES, LATERAL POSITION)

	CASES	PLEURA OPEN (MIN.)	pH	SERUM CO_2 (MM./L.)	CO_2 TENSION (MM. HG)
Gibbon et al. (Machine)	13		7.22	26.8	74
Taylor and Roos (Manual)	6	105	7.21	25.7	67
Beecher et al. (Manual)	8	142	7.26	26.6	58

by the simple bag squeezing method such as we used is at least as good and apparently somewhat better and more dependable than that by machine, and that the prone and possibly supine positions are better than either.

SUMMARY

Work by others has confirmed our original finding that carbon dioxide accumulates to dangerous levels during surgery in the open thorax. This accumulation certainly is related to position of the patient when these patients have an open pleura, it is due to impairment of respiration, of ventilation, of the lungs. It can be, if not entirely eliminated, at least so minimized as to make it of no consequence by nothing more than choice of a favorable position. On the other hand, the simple expedient of bag squeezing by hand to assist the respiration gives dependable and relatively safe protection, at least as good as and apparently better than artificial ventilation by the more complex machine.

The prone and the supine positions are most favorable for the natural excretion of carbon dioxide, but let us say once again it is not our view that surgeons operating in the thorax need adopt either the prone or the supine position. They can choose whatever position that best suits them and suits best the surgery to be done, and rest confident that simple bag squeezing by hand will protect their patient, if they choose the lateral position.

In conclusion, we have shown the existence in thoracic surgery of a real and a common danger. This has been confirmed by others, and we have shown that it can be overcome or at least effectively minimized by simple means. The use of these simple measures is extremely important.

We wish to acknowledge our indebtedness to Dr. Richard H. Overholt of the Deaconess Hospital, Boston, and to Dr. Edward D. Churchill and Dr. Richard H. Sweet, of the Massachusetts General Hospital, Boston, for their permission to study blood gases in their cases.

REFERENCES

1. Beecher, H. K.: Severe Acidosis Associated With Trans-Thoracic Surgery, N. I. H. Bull., January, 1950.
2. Beecher, H. K., Francis, L., and Anfinsen, C. B.: Acid-Base Balance During Ether Anesthesia, J. Pharmacol. & Exper. Therap. **98**: 38, 1950.
3. Beecher, H. K., and Murphy, A. J.: Acidosis During Thoracic Surgery, J. THORACIC SURG. **19**: 50, 1950.

4. Bunker, J. P., and Beecher, H. K.: Metabolic Changes During Anesthesia in Poor Risk Patients. In preparation.
5. Etsten, B.: Personal communication.
6. Gibbon, J. H., Jr., Allbritten, F. F., Jr., Stayman, J. W., Jr., and Judd, J. M.: A Clinical Study of Respiratory Exchange During Prolonged Operations With an Open Thorax, Ann. Surg. 132: 611, 1950.
7. Maier, H. C.: Personal communication.
8. Miller, F. A., Hemingway, A., Knight, R. T., Nier, A. O., and Varco, R. L.: The Evaluation of Respiratory Abnormalities by Means of a Portable Mass Spectrometer, N. I. H. Bull., January, 1951.
9. Taylor, F. H., and Roos, A.: Disturbances in Acid Base Equilibrium During Ether Anesthesia, J. THORACIC SURG. 20: 289, 1950.

DISCUSSION

DR. H. C. MAIER, New York.—Dr. Beecher stated, I believe, that in their series acidosis occurred only during thoracic operations, especially in the lateral position. We, however, have even encountered acidosis in some abdominal cases operated upon in the supine position with ether anesthesia. One can naturally say that this is because the anesthetist was not adequately ventilating the lungs, and such criticism is justified. The point I wish to stress, however, is that it is my impression that as ether anesthesia is employed throughout the country there are many instances in many clinics, the same as in ours, where acidosis occurs due to depressed respirations. Of course the problem of respiratory acidosis is a much more important one during thoracic procedures with an open chest.

(Slide.) This first slide indicates the pH and CO_2 tension changes in a patient undergoing lobectomy and decortication in the lateral position. I would like to emphasize at this point that we feel strongly that every anesthetist, every anesthetic technique, as well as the anesthetic agents, may cause certain variations. In this patient, as is not infrequently true in our cases, we tended to get the patient fairly deep before intubation was done in order to have it performed in a smooth manner from a technical standpoint. As a result in this case we had respiratory acidosis early, although the oxygen saturation stayed quite normal throughout operation. So here we have an instance in which before the patient was turned in the lateral position we had a lower level of the pH than at any time throughout the intrapleural part of the operation. This shows the variations that may occur.

(Slide.) This chart records the pH and CO_2 changes during an operation for mediastinal lymphosarcoma performed through a lateral approach in a somewhat emphysematous individual. Here again we had a marked drop in pH before we turned the patient into the lateral position, due to decreased ventilation, and in this patient too there was no hypoxia at any time. Then the anesthetist gave more adequate ventilation while in the lateral position and corrected the respiratory acidosis, but then another drop in pH occurred later due to the combination of open chest, lateral position, emphysema, and less adequate ventilation. Rapid improvement in the acidosis occurred at the end of the operation in this patient who had no secondary circulatory disturbance.

(Slide.) Here is a case of abdominal gastrectomy with ether anesthesia showing a drop in pH. One can correctly say that this acidosis is due to depressed respirations and inadequate ventilation. But the point is that it did occur in this patient, although there was apparently clinically satisfactory anesthesia with normal oxygen saturation throughout.

(Slide.) This slide shows the pH plotted against length of time from the beginning to the end of a miscellaneous group of operations. You will note that in the absence of any circulatory disturbance occurring during operation there is a rather rapid correction of respiratory acidosis at the end of the operation. Determinations made only at the beginning and the end of operation may give us very little information about what may occur in between these times. If we study this problem we have to get repeated readings throughout the procedure.

DR. EVARTS A. GRAHAM, St. Louis.—I had no intention whatever of discussing this paper when I came here, but I would like to call attention to one or two very simple things. We are constructed in such a way that the act of inspiration consists, at least partly, of elevation of our ribs and expansion of the thorax forward by that means. If we lie on the chest in the prone position without any assistance in getting air into the lungs, with

each inspiration we have to raise the trunk off from whatever it is we are lying on. In other words, our respiratory muscles have to do much harder work than they had to do before. If we lie on one side we can breathe more comfortably than if we lie flat on the chest, and measurements of vital capacity in a normal individual will show that fact. However, if, with the patient in the lateral position, one opens one side of the chest, and puts that part of the chest out of commission more or less as a respiratory organ, then the unopened side of the thorax has to lift the trunk with each inspiration, and is handicapped by not having the help of its fellow on the other side, and so of course without artificial assistance again respirations are going to be seriously embarrassed. If we lie on the back, all we have to do with inspiration is to enlarge the front part of the thorax; we do not have to lift the trunk with each inspiration. Therefore, from the standpoint of an ideal position to minimize handicaps to inspiration, it naturally follows that the best is on the back—the supine position. The conditions are still further improved, as judged by the vital capacity, if the trunk and head are elevated to an angle of about 45 degrees. In any condition, however, in which respiration is embarrassed by fluid, by weak muscles, low vital capacity, anesthesia—whatever you wish—it is natural that we ought to protect the body as much as possible by giving artificial assistance.

It seems to me that much of this discussion about whether or not a patient should be operated on in the lateral position, supine position, or prone position, comes down chiefly to the question of—are you going to see to it that the patient ventilates his lungs properly? That is really the problem.

During our work on the Empyema Commission in World War I we did many experiments on ourselves, that is, on normal people and on sick people as well, and we thought that we demonstrated pretty satisfactorily that the prone position, of all of them, is the worst from the standpoint of ventilating the lungs automatically, spontaneously, if the patient is lying on a hard surface, because, as I have just said, with each inspiration he has to lift his whole trunk every time he breathes. Of course Dr. Overholt's table makes the problem a different proposition; it is not the same thing at all. But it all gets back to the fundamental proposition of ventilation, and I think position is much less important than to bear in mind the fact that it is ventilation that is needed.

DR. PAUL W. SANGER, Charlotte, N. C.—I think Dr. Beecher has shown convincing evidence that there is an increased CO_2 tension with the patient in the lateral position, and the reason we do not get into trouble more often must be because the safety factor is pretty large. There are a few conditions, however, in which we cannot depend on the safety factor. We think there are four such conditions, namely: (1) when there is a case with anticipated large blood loss when it is recognized beforehand that the lung is adherent to the chest wall; (2) when the patient is acutely ill; (3) when the vital capacity is low, especially in emphysematous patients; and (4) when we cannot depend on the anesthesia.

In these four groups of patients we are obliged to operate with the patient in the supine position. We have found it expedient to do an anterior thoracoplasty with the removal of the first five ribs to the posterior axillary line, also including their respective costal cartilages. By using the anterior approach, after having removed the ribs, we find that the mediastinal structures can be isolated, ligated, and severed very easily before extreme hemorrhage is created by avulsing the adherent lung from the chest wall. Thereupon the bleeding can be readily controlled by pressure. The postoperative emphysema problem, which has been discussed this morning, we feel is adequately controlled by the anterior removal of the first five ribs. The mere position of the patient reduces the contralateral infection factor, and we think the partial thoracoplasty minimizes the postoperative formation of bronchopleural fistula and empyema. In the seventeen patients on whom we have used this procedure, we have had no symptoms suggestive of an increased CO_2 tension syndrome or other uncontrollable complications.

DR. RICHARD H. OVERHOLT, Brookline, Mass.—I think we will all agree that the work of Dr. Beecher has contributed to our total knowledge of the problem of how best to maintain ventilation during intrathoracic procedures. Dr. Graham mentioned a very important point which was, in effect, that although laboratory studies do seem to indicate that one

position might favor another, when we are confronted with the problem of getting the sick person well each surgical team should select that type of approach which will provide the best operation in its hands. There is an advantage for a particular surgical team to develop a more or less routine type of approach that can be carried out with dispatch and convenience. In that way, you add to the safety factor.

Originally, in working with the prone position, we had in mind improving the drainage from the lung as well as improving ventilation on the contralateral side. Then we found that the most serious-risk patient, the patient with the wettest lung, tolerated the open chest better in that position than the good-risk patient with a dry lung did in the lateral position. We came to the conclusion that if this position was of advantage to a bad-risk case, it ought to add to the safety in the management of the good-risk case. Another advantage of the prone position is that one may want to do something more than just excise a lung and, in the supine position, it is a little difficult to carry out a concomitant thoracoplasty if a simultaneous thoracoplasty is contemplated at the time of resection. Again, in some instances where the lung is densely bound down or is involved in a large tumor mass, it is important to have a wide exposure. Then, an approach from the posterolateral aspect has great advantage. There are many factors which enter into the equation, and no one will quarrel with the general proposition that each surgical team must select that particular position which will provide for its patient the greatest margin of safety.

DR. JOHN H. GIBBON, Jr., Philadelphia.—I think we are all agreed that when the pleura is open ventilation is very important. I think it would be nice to state before this Association that our Honorary Member, Clarence Crafoord, was one of the first, ten years or more ago, to emphasize the importance of pulmonary ventilation under these conditions. Also, Drs. Beck and Mautz were among the first thoracic surgeons in this country to appreciate Crafoord's work.

I think Dr. Beecher is to be congratulated upon being the first to report a carefully studied series of patients demonstrating the danger and the everpresent hazard of accumulation of CO_2, with an increase of CO_2 tension and a drop in pH. I think he has also demonstrated that it is probably more difficult to provide adequate ventilation in the lateral position. However, I am not yet convinced that we cannot provide adequate ventilation in that position.

Another thing which I think should be borne in mind is that with these relatively small series of cases which we all have, there is quite a large individual variation. Both Dr. Beecher and I have reported averages and have also given individual cases. But we have had patients, as Dr. Beecher has I know, that have been operated upon in the lateral position without any respiratory acidosis.

Why is it that is some cases acidosis develops in spite of apparently adequate ventilation? I think very often it is because the patient is emphysematous or has a greatly reduced vital capacity. In other words, we are dealing with the type of patient in whom, probably in any situation, it might be difficult to avoid an increase in CO_2 tension. I think before we come to rigid conclusions about position or method of ventilation—I hold no brief for the mechanical method of ventilation—we should remember that our series are still small, that we must collect more data, and that we must ascertain the facts about these enormous individual variations.

Finally, I might add relative to that point that we did study one patient on whom a pneumonectomy was performed in the Overholt face-down position. This patient had the most severe respiratory acidosis of any in our series despite determined effort to provide adequate ventilation. The citing of this single case again does not constitute evidence that the position was at fault. I think Dr. Beecher has very suggestive evidence that in the prone and supine positions it is easier to avoid respiratory acidosis than it is in the lateral position. I am merely pointing out that there are marked individual variations with the use of any position.

What we all want to be able to do is to recognize the development of respiratory acidosis when the patient is being operated upon. We do intermittent blood sampling now, which necessitates maintaining an indwelling needle in an artery throughout the operation. The sample has to be analyzed, which takes time, and you do not know during the course of the operation whether or not the CO_2 tension is going up and the pH dropping. I think we

are all interested in the development of some method of knowing quickly, easily, and continuously the percentage of carbon dioxide in the expired gases during a prolonged operation. I hope someone will develop such a method in the next few years.

DR. LLOYD H. MOUSEL, Seattle.—Dr. Beecher is to be complimented for the fine work he has done on respiratory acidosis.

He spoke about assisted respiration. Perhaps we should mention some of the methods of assisting respiration and compare them with controlled respiration. There is a great deal of difference, and certainly we must at times interfere seriously with the lesser circulation when we assist or control this most important function.

In a preliminary study made by Dr. Howard Pierpont a little more than a year ago, pressure readings were made within the pulmonary arteries of dogs during assisted and controlled respiration. The pressure within the pulmonary artery rose markedly in those animals which were being subjected to controlled respiration. In my opinion, Pierpont's observations are of clinical importance. If we control respiration, or assist it too vigorously, it seems reasonable to assume that a pulmonary hypertension will develop. This increased work load, suddenly thrown upon a right heart in which myocardial changes are already present, may result in congestive heart failure or even sudden death during operation. Young patients with normal hearts undoubtedly compensate for the pulmonary hypertension which results from positive pressure breathing. Patients in the older age group ought to be watched carefully. If assisted respiration is necessary the mechanical efforts need to be coordinated with the patient's own efforts to breathe. The amount of pressure exerted on the breathing bag should be limited to that pressure which will assure a sufficient tidal volume of respiration. At the end of the inspiratory phase the pressure within the breathing system should be allowed to drop to atmospheric pressure.

Controlled respiration should be avoided if possible. In case there is need to control respiration for a short period, sustained pressures are to be avoided. Any amount of constant positive pressure within the breathing system will be transmitted to the alveoli. The pulmonary capillary system will be compressed, preventing adequate filling and resulting in increased pressures within the chambers of the right side of the heart.

DR. OSLER A. ABBOTT, Atlanta.—First, I would like to say that the ventilation factor so thoroughly stressed by Dr. Beecher is probably the most important one in this discussion. I wonder if, by any chance, we are missing something else of major importance. There is a difference in the position, both of the lobe upon which we must operate and the adjacent lung structures, according to the position in which the patient is operated upon. The degree of traction upon an adjacent lobe and displacement may well kink the vessels in the hilus, and in some cases partially obstruct pulmonary venous return and set us a temporary state of pulmonary vascular congestion. This kinking is much minimized by the face-down position. There is also the factor of the traction and torsion upon the bronchial hilar structures and their effect upon pulmonary function and bronchospasm.

We used to be members of the lateral position school. It was customary, during the first week or ten days following surgery, to visualize a well aerated residual lobe following lobectomy, but to find rather inadequate breath sounds on auscultation. Following cough these breath sounds would come through in a mildly tubular character. We have felt that this is a good clinical sign of pulmonary dysfunction on the operated side and may be related to resolution of congestion, or even of ecchymosis in the retracted lobe. This situation, of diminished to absent breath sounds over a residual, roentgenologically demonstrably expanded lobe, does not occur following the face-down position, and we feel it is due to the lack of torsion upon the hilar pedicle and, indeed, the lack of handling this lobe almost completely during surgery. We would therefore like to stress that there is more than just the ventilatory factor; there is also the handling of the lobe and the kinking of the vascular pedicle and of the bronchial pedicle to the lung.

Before terminating my remarks, I would like to emphasize the danger of the position of the sternal rest which may be used in the face-down position, particularly with some of the attachments which are used with the usual operating table. The lower sternum may be a remarkably flexible structure and, in a patient with a thin P-A diameter of the chest, there

may be a very marked compression of the heart and actually a physiologic cardiac tamponade produced if the sternal rest is placed in the midsternal or lower sternal region. It is very important that this rest be placed so that it impinges upon the sternal notch. This is a good thing for which to look in the patient whose blood pressure falls and pulse pressure seems to contract when placed in the face-down position.

DR. ROBERT H. WYLIE, New York.—Added evidence of this mechanism has been observed by us during the course of operation for mitral stenosis. With continuous electrocardiography we have observed bundle of His and nodal rhythm, and T-wave changes which have been completely obliterated by asking the anesthetist to increase the ventilation.

DR. CLARENCE DENNIS, Minneapolis.—Some of us in Dr. Wangensteen's department in Minneapolis have been working for several years to try to develop a combined pump and oxygenator that would support the entire circulation without the employment of the lungs or heart of the subject in question. One of the major problems which has perplexed us is the problem of elimination of carbon dioxide in our dogs prior to the time the machine is employed. We have had our dogs on one side much of the time and have opened both sides of the pleura, and it has been possible to examine the down side lung, both during and after the procedure, and in those dogs sacrificed some time afterward.

We were not satisfied with any of the methods of maintaining respiration, using the lungs, that were available to us. We therefore developed a respirator which seems to have solved the problem for us. This is not the time to describe this, but I would like to point out that the things that seemed important were these: In the first place, the expiratory phase must be characterized by a sharp drop all the way to atmospheric pressure, as Dr. Mousel emphasized. In the second place, during the inspiratory phase, the lung must be maintained with a plateau rather than a peak of pressure, and we have found that, so far as our dogs were concerned, a plateau of 16 or 17 cm. of water pressure occupying about one-third of the respiratory cycle is enough to keep the down side lung from undergoing compression atelectasis from the weight of the mediastinal structures that lie on the lung during the time the dog is in that position.

DR. BEECHER (closing).—I am grateful to Dr. Graham, Dr. Maier, Dr. Sanger, Dr. Overholt, Dr. Gibbon, Dr. Mousel, Dr. Abbott, Dr. Wylie, and Dr. Dennis for their helpful discussions.

Dr. Maier emphasized the fact that too deep anesthesia is undoubtedly common over the country; I know that is so. One need not use overly deep anesthesia to carry out any kind of surgery needed. Of course the most difficult problem so far as this is concerned is upper abdominal surgery. We have a good deal of blood gas data during upper abdominal surgery and we know that abnormal elevation of carbon dioxide tension is not necessary even here. The acidosis Dr. Maier spoke of midway in his operations is doubtless due to the fact that most anesthetists (and surgeons) like to have their patients much more deeply anesthetized midway in the operation than they do at the end. We hope we have shown that the surgeon operating in the thorax can use whichever position is best for the surgery and best for his own practices. It is obvious that if the surgeon does use the lateral position, his anesthetist must employ some kind of aid to ventilation. I hope I did not sound dogmatic in arguing for the bag-squeeze method over the machine. I am simply recording that, at the present time, the simple bag-squeeze method—and I must say simple methods appeal to me more than complex ones—the simple educated hand is at least as good, so far as we can tell from factual data at present available, as the more complex machine. As Dr. Gibbon pointed out, four of his pneumonectomy patients did show acidosis. Neither are we successful in maintaining the acid-base equilibrium we would like in the pneumonectomy case. Neither method is perfect. We need to know a good deal more about how to perfect these methods. Rises in four out of thirteen is not good enough. Our record is no better and it is not good enough either. The situation could be handled much better if we had some way, as Dr. Gibbon pointed out so well, of detecting when the pCO_2 rises to troublesome levels. Dr. Wylie spoke of his work with Dr. Himmelstein indicating that electrocardiograms are of assistance, and perhaps that will be one way of indicating when the tension is getting too high.

MUCOID IMPACTION OF THE BRONCHI

ROBERT R. SHAW, M.D.*
DALLAS, TEXAS

WITHIN the past four years we have seen ten patients suffering from what seems to be a distinct clinical entity within the lungs that has not been clearly described in medical literature. These patients all had an underlying condition of asthma or chronic obstructive bronchitis. We have used the descriptive term "mucoid impaction of the bronchi" to designate this entity. It is important that it be recognized clinically since its symptomatology and roentgen appearance may closely simulate other diseases—namely, neoplasm, abscess, and tuberculosis—which have an entirely different prognosis.

Mucoid impaction of the bronchi results from a localized accumulation of inspissated mucus in the bronchi. Typically, this impaction occurs in the second order branch bronchi distal to a bifurcation. The impaction grows in size, layer by layer, in a laminated manner to finally dilate the bronchus in which it lies to many times its normal diameter. As the impaction enlarges it compresses the neighboring branch bronchus so that it also accumulates a mucoid mass which enlarges in the same manner. As the bronchial wall is dilated by the mucoid mass it becomes infected. The cartilaginous elements are almost entirely destroyed, and there is an infiltration of lymphocytes and eosinophilic neutrophils into the wall. The characteristic yellowish-white appearance of the normal bronchus changes to a reddish brown. The mucous lining loses its ciliated columnar epithelium and is replaced by squamous epithelium. Beyond the blocked bronchus suppuration occurs. This suppuration may be in the form of an abscess. This abscess, however, is of the nonputrid variety. At times the suppuration is entirely intrabronchial, causing eventually the formation of cystic bronchiectasis. Typically these cysts will be completely filled with purulent secretion. Seldom will air slip by the mucoid masses to occupy space within these cysts. The alveolar spaces which remain intact contain an amorphous eosinophilic staining material which contains numerous plasma cells, lymphocytes, and occasionally giant cells. Commonly the bronchioles are filled with neutrophils and lipophages. Fat stains may reveal fat in epithelial cells of some of the bronchioles and alveoli, and at times the fat is free due to the breakdown of the macrophages.

The plugs of mucus range from 2.5 to 3.5 cm. in length and from 0.9 to 2.3 cm. in diameter. They are a glary, greenish-gray in appearance, and putty-like in consistency. The cut surface reveals a thick, tenacious ropy material. Section of the plugs reveals evidence of lamination.

From the Surgical Service of the Southwestern Medical College of the University of Texas.

Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

*Clinical Professor of Surgery, Southwestern Medical College.

The most striking feature of the pathology is the gross finding of the paired impactions of mucoid material in major branch bronchi, and the lipoid granulomatous inflammation of the destroyed wall of the involved bronchus and the pulmonary tissue beyond the plugs.

ROENTGEN FINDINGS

The roentgen appearance of mucoid impaction may be quite varied. The mucoid plugs themselves are large enough to be shadow producing (Figs. 4 *A* and 5 *A*). The shadow is characteristically near the hilum of the lung and appears as a V-shaped shadow. As with a hilar neoplasm the shadow cast by the plugs may be masked by the larger shadow due to the suppuration beyond (Fig. 3 *A*). The shadow cast by the suppuration varies. It may be typical of a pneumonitis (Fig. 2 *B*). This may be the reaction actually present in Loeffler's syndrome. Characteristically the suppuration beyond the plugs casts a sharply circumscribed dense shadow which is indistinguishable in appearance from a pulmonary neoplasm (Figs. 2 *A*, 3 *A*, and 6 *A*). If air is able to slip past the mucoid plugs an annular shadow denoting an abscess with a fluid level may be seen (Fig. 7 *B*). In other instances cysts uniform in size with or without fluid levels may be evident (Fig. 5 *A*). Serial roentgenograms may show clearing of one segment of lung only to have the same type of shadow reappear in other distant segments. At times the sharply circumscribed shadows may completely disappear leaving little roentgen evidence of damage to the lung (Fig. 7 *D*). With clearing of the suppuration the shadows cast by the plugs may persist.

CASE REPORTS

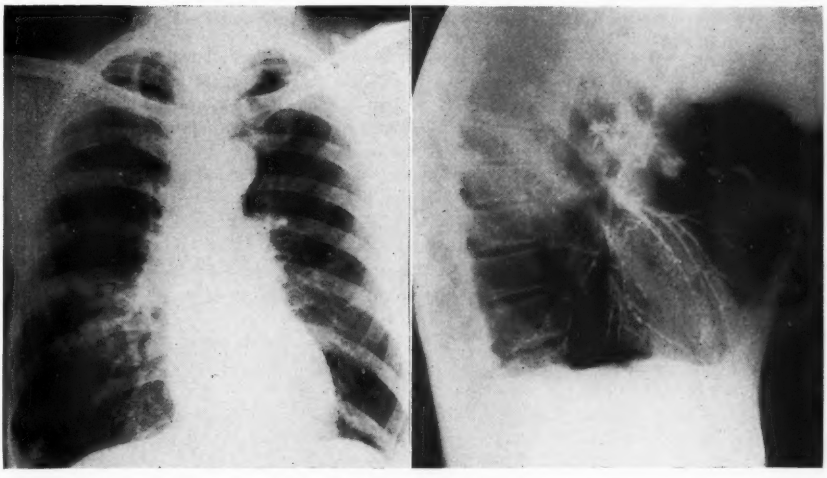
In eight of the ten cases being reported the diagnosis was confirmed by examination of the surgical specimen.

CASE 1.—E. C. F., a 54-year-old man, had had frequent intermittent colds and cough for the past twenty-five years. The cough had always been nonproductive. For the past nine months he noticed dyspnea on exertion, weakness, and anorexia. There had been no fever.

Bronchoscopy: (May 11, 1948.) There was thin watery secretion in the trachea, both bronchi, and their branches. Around the dorsal bronchial orifice on the right the mucosa was thickened and inflamed. Purulent material could be seen coming from this bronchus. No neoplastic tissue could be seen. Bronchography showed obstruction of the dorsal bronchus on the right, and cystic bronchiectasis of the anterior segment of the right upper lobe (Fig. 1*B*).

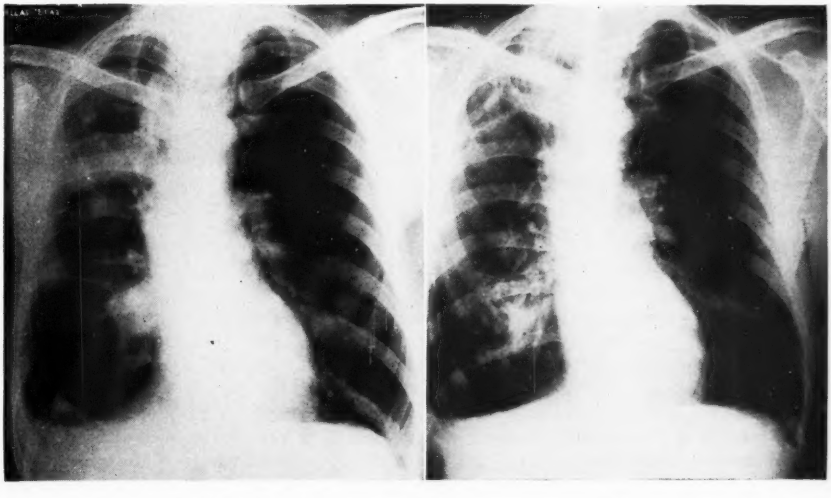
Operation: (June 24, 1948.) Preoperative Diagnosis: Question of bronchiogenic carcinoma. Bronchiectasis anterior segment, right upper lobe. The right chest was opened through the sixth rib bed. There were adhesions between the dorsal lobe and the posterior chest wall and also adhesions in the interlobar fissures, but otherwise the pleural cavity was not obliterated. A hard mass could be felt in the dorsal lobe which was thought to be carcinoma. The bronchus to this lobe was especially hard, and there was no doubt in the operator's mind that a malignancy was present. The dorsal lobe was removed by dissection technique. The anterior inferior segment of the upper lobe was removed in the same manner. Before the chest was closed the specimen was examined, nad it was found that two large plugs of inspissated mucus had greatly dilated the branch bronchi to the dorsal segment. Beyond these dilated bronchi was saccular bronchiectasis with pneumonitis and fibrosis.

Postoperative Course: In the first postoperative check-up film a rounded shadow approximately 4 cm. in diameter was noted in the lower portion of the right chest. (Fig. 1*C*.)



A. *B.*

Fig. 1, *A* and *B*.—(Case 1) Note diffuse shadow in right hilum. Lateral bronchogram shows saccular bronchiectasis of anterior segment of upper lobe and abrupt block of dorsal bronchus by a discrete shadow-producing lesion. Block was mucoid impaction.



C. *D.*

Fig. 1, *C* and *D*.—Two months following resection of anterior segment of right upper lobe and dorsal segment of lower. A rounded shadow due to mucoid impaction has appeared which later almost completely disappeared.

The etiology of this shadow was not determined, and one month later it had disappeared. (Fig. 1*D*.) The patient continued to have cough and expectoration of moderate amounts of thick, tenacious mucus. He did, however, begin to gain weight and strength.

CASE 2.—J. L. B., a 25-year-old physician, had had bronchial asthma all of his life. Skin tests had revealed numerous specific allergies. A roentgenogram of his chest in April, 1947, was perfectly clear. In June, 1947, he had a bout of fever which was diagnosed as atypical pneumonia. At this time in the roentgenogram a shadow was seen in the base of the right upper lobe. He responded well to penicillin, but the shadow in the subapical segment of the right upper lobe never entirely disappeared. This was observed over a period of a year. Since the shadow persisted, and the patient continued to have low-grade fever, multiple hemoptyses, and undue fatigue, right upper lobe lobectomy was advised.

At operation, July 16, 1948, examination of the right upper lobe bronchus revealed gross dilatation of the branch bronchi to the subapical segment of this lobe. Since the bronchus was partially destroyed it was decided that an upper lobe lobectomy would be necessary in order to section the bronchus in normal tissue.

Pathologic Examination: Upon opening the grossly dilated bronchi two large plugs of inelastic soggy, dessicated, gellatinous mucus were found. Beyond the plugged bronchi was cystic bronchiectasis filled with creamy pus.

Postoperative Course: The patient made an uneventful postoperative recovery and stated that he has been more free of the asthma than at any other period in his life.

CASE 3.—E. Z., aged 14 years. One month before admission to Baylor Hospital on Aug. 2, 1947, this boy noticed a slight cough and fever. The cough was nonproductive. The temperature rose to 103° F. He was treated with penicillin, and the temperature soon receded to normal. The roentgenogram of the chest showed a shadow in the right upper lobe which had persisted in spite of the chemotherapy. There was no sputum. This patient had been asthmatic all of his life. On August 12 a right upper lobe lobectomy was done. At surgery, there were found to be dense adhesions between the visceral and parietal pleura over the right upper lobe. There were numerous enlarged lymph nodes in the hilum of the upper lobe.

His convalescence was uneventful, and he has remained well.

Pathology: There was marked induration and firmness throughout 85 per cent of the specimen. On cut surface through the indurated area there was a marked outflow of thick, purulent material from numerous areas. On tracing the bronchi a large impaction of mucoid material was encountered measuring approximately 3½ cm. in length and 1.3 cm. in diameter. This mucoid mass was blocking the bronchus to a major portion of the lobe.

CASE 4.—J. T., aged 45 years, had been troubled with asthma most of his life. He was a painter by trade. Because of fever and malaise a roentgenogram was taken of the chest on April 22, 1949, which showed a dense area in the right upper lobe. (Fig. 2*A*.)

Bronchoscopy: (Nov. 9, 1949.) The mucosa surrounding the upper lobe orifice was hyperemic and edematous. There was no evidence of neoplasm. A sucker tip was passed into the right upper lobe bronchus and a small amount of thick secretion was obtained which was studied for neoplastic cells. This study revealed no atypical cells.

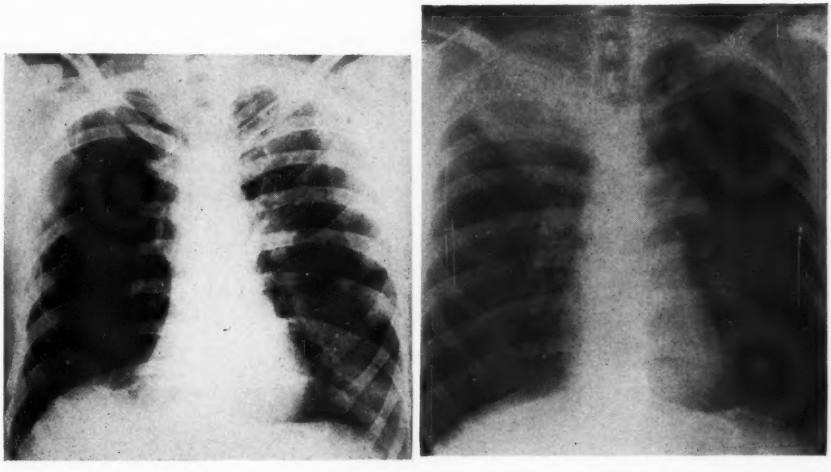
Preoperative Diagnosis: Nonputrid abscess of lung? Bronchiogenic carcinoma.

Operation: (Nov. 10, 1949.) The upper lobe was removed without difficulty by ligation technique. A complete parasympathetic denervation of the remaining portion of the right lung was carried out because of the underlying asthmatic condition.

Pathology: The lobe was completely replaced by a spherical mass measuring 10 cm. in diameter. On cross section multiple cavities all filled with greenish purulent material were noted. The major bronchi to the lobe were completely filled and dilated by large, gummy plugs of mucus.

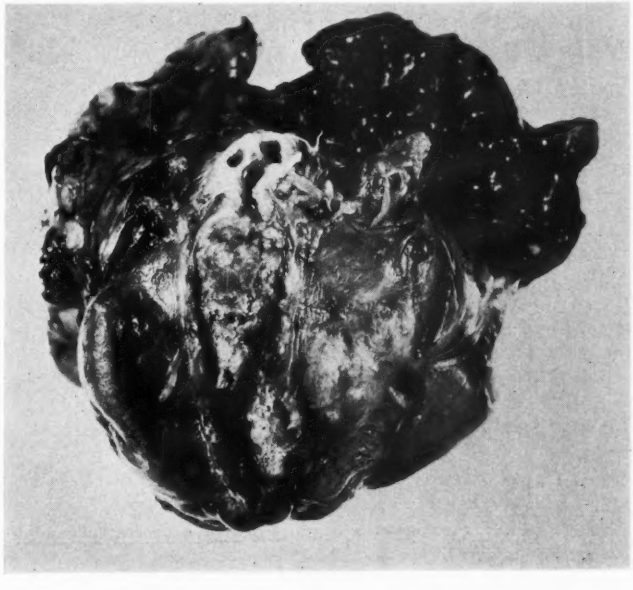
Postoperative Course: The patient made an uneventful recovery from the surgery and returned home. At check-up examination on December 17, it was noted that he was coughing very little. He had gained weight, and the breath sounds seemed clearer on the right than on the left side. In April, 1950, however, he began to have further trouble with

his chest. At that time, roentgenogram showed areas of pneumonitis in both the upper and lower lobes on the left, which indicated that the same process that had been seen in the right upper lobe was starting there (Fig. 2*B*).



A. *B.*

Fig. 2.—(Case 4) *A*, Dense shadow in right upper lobe. Upper lobe lobectomy revealed mucoid impaction with nonputrid abscess, beyond obstructed bronchus. *B*, Six months following lobectomy there was an indication of the same process beginning in left lower and left upper lobes.



C.

Fig. 2, *C*.—(Case 4, J. T.) Right upper lobe. Note large ovoid impaction of mucus dilating and blocking a branch bronchus.

CASE 5.—Mrs. J. M. was admitted Feb. 15, 1949. This 57-year-old woman had noticed a pain in the left chest which radiated down the left arm for the past year. There had been no cough and no weight loss. She stated that she wheezed when she lay on her left side. She had noticed no fever. The past history was noncontributory except for a questionable coronary attack seven years previously.

The roentgenogram of the chest showed a large, rounded shadow in the left midlung field (Fig. 3*A*).

A diagnosis of pulmonary neoplasm was made, and surgery was advised.

Operation: (Feb. 16, 1949.) When the left chest was opened the upper lobe was found to be densely adherent to the chest wall. The lower lobe was completely free of adhesions. Since the hilum was uninvolved, only the upper lobe was removed.

The patient's convalescence was uneventful, and she has had no recurrence of similar trouble.

Pathology: Upon sectioning the lobe removed, a large multiloculated space was opened which contained brownish semisolid material. The bronchus supplying this area was greatly dilated by a large mass of inspissated mucoid material. Microscopic examination of this bronchus showed the mucosa to be thin and atrophic and densely infiltrated with inflammatory cells.

A. *B.*

Fig. 3.—(Case 5) Left upper lobe lobectomy done for suspected neoplasm. *A*, Mucoid impaction found. *B*, Eight months following resection chest was clear.

CASE 6.—Mrs. J. B. H., aged 53 years, developed virus pneumonia in March of 1949. She had a hard unproductive cough. She stated that she had had previous difficulty with her right lung. She had been asthmatic all of her life. At one time she coughed up a hard plug of material, and this was followed by the coughing up of much pus. Roentgenograms showed destruction of the right middle lobe (Figs. 4*A*, *B*, *C*). Right middle lobectomy was devised.

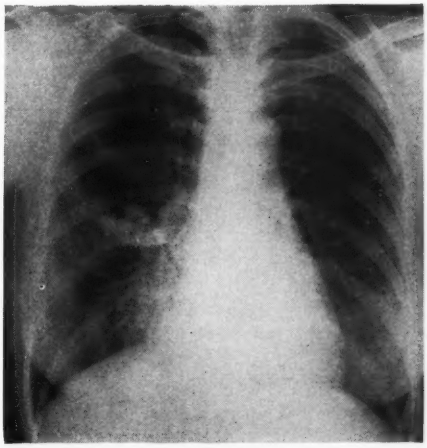
Preoperative Diagnosis: Mucoid impaction of the right middle lobe bronchi with residual fibrosis.

Operation: The pleural cavity was free of adhesions except over the middle lobe where adhesions were dense and vascular. The middle lobe was removed by dissection technique.

Postoperative Course: Postoperative convalescence was uneventful, and the patient's health has been greatly improved.

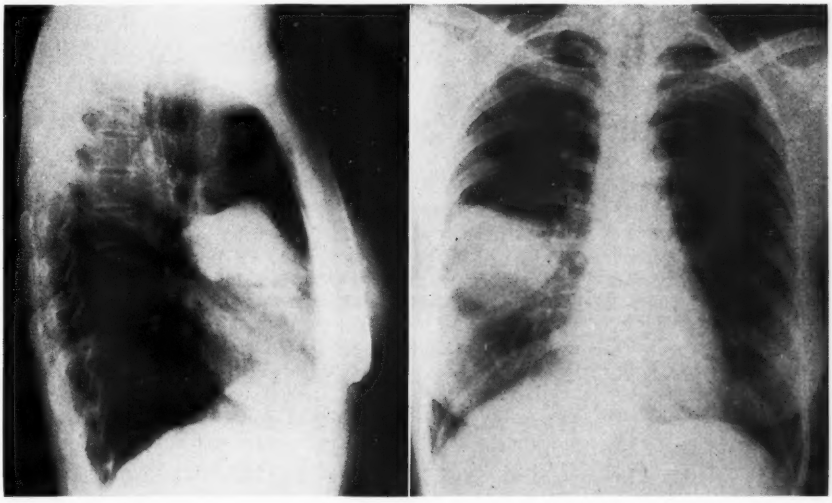
Pathology: One branch bronchus of the middle lobe contained a large, oval foreign body of thick, tenacious, mucoid material. It measured 2½ cm. in length by 0.9 cm. in

diameter. Sections through other areas revealed similarly dilated bronchi which extended toward the periphery of the lung tissue. These were filled with tenacious mucoid material. Beyond these areas there was bronchiectasis, cylindrical in type. Sections through the gross mucous plug obstructing the bronchus revealed it to be composed of laminated layers of eosinophilic mucoid material.



A.

Fig. 4, *A.*—(Case 6) Note shadows in right hilum cast by mucoid masses dilating the middle lobe bronchi.

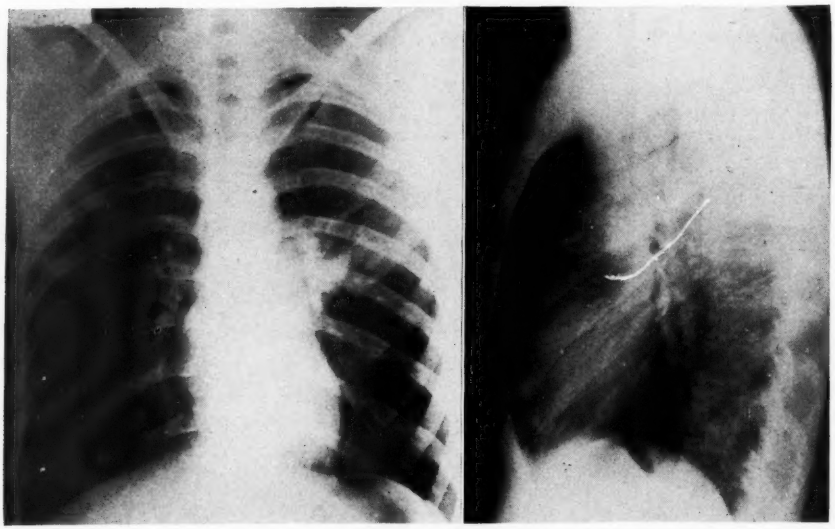


B. *C.*

Fig. 4, *B* and *C.*—Same patient five months later. The middle lobe was swollen due to suppuration beyond the obstructed bronchi.

CASE 7.—O. B., aged 19 years, had been sick for over a year with a chronic cough which was productive of small amounts of purulent sputum. Tubercle bacilli could never be found in the sputum. Nevertheless a diagnosis of pulmonary tuberculosis was made, and he spent six months in a tuberculosis sanatorium. It was finally realized that his disease probably was not tuberculosis. Since this patient had been troubled with asthma most of his life, it was suspected that he had mucoid impaction of the bronchi. Exploration of the left chest was advised.

Operation: (March 2, 1950.) The upper lobe was found to be adherent to the anterior chest wall over a rather extensive indurated area. It could be seen that a portion of the indurated area was definitely cystic. The upper lobe was removed.



A *B.*

Fig. 5, *A* and *B*.—(Case 7) The lift hilar shadows were due to mucoid impaction. The peripheral shadows proved to be pus-filled bronchiectatic cysts.

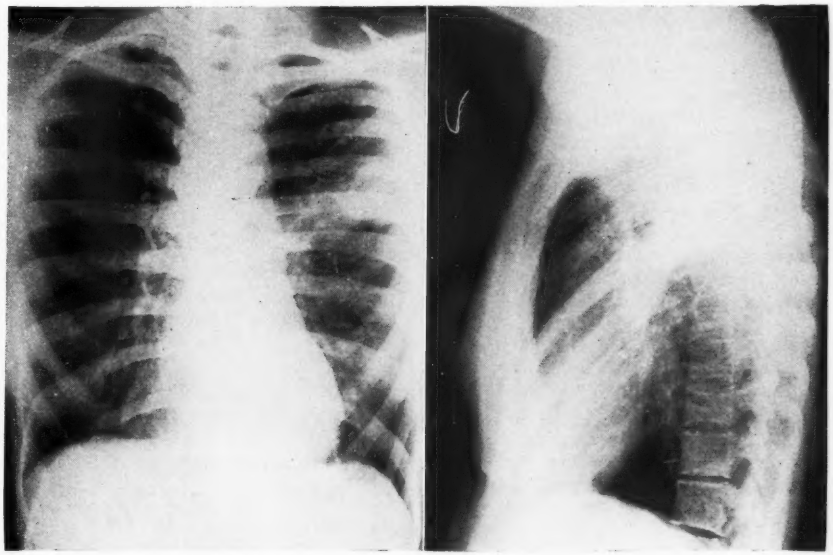
Postoperative Course: The postoperative course was marked by excessive bleeding from the parietal pleura which necessitated returning the patient to the operating room for evacuation of a large clot in the pleural space. Following this he made a good postoperative recovery. He was seen again in November, 1950. At this time he stated that he was beginning to have fever again and was not feeling well. A roenegenogram of the chest showed that there was pneumonitis in the right upper lobe, suggesting that the same process was again beginning in a segment of this lobe.

Pathology: Evidence of disease was limited to the posterior and apical segments. The lingula was normal. In the bronchi to these segments there was impaction of thick mucoid material so that the bronchi were dilated to more than three times normal caliber. Beyond these dilated bronchi were cystic areas completely lined with epithelium. These cysts were filled with thick, creamy, mucopurulent material. On microscopic section, there were some areas of the lung near the region of the encapsulated cystic spaces where there were seen a great number of lipophages.

CASE 8.—J. R., a 26-year-old woman, had been an asthmatic since childhood. Her mother had been a chronic asthmatic, and several members of the mother's family had been confirmed asthmatics. At the age of 7 years she had extensive skin testing done to determine a possible

allergic background. In 1949, a sharply circumscribed shadow was noticed in the left upper lung field posteriorly (Fig. 6*A* and *B*). This shadow did not change in size or shape over a period of months of observation.

Because of the underlying history of asthma, the general good condition of the patient, and the appearance of the roentgenogram, a diagnosis of mucoid impaction of the bronchi was made in October, 1949. Surgery was not recommended. Due to the fact, however, that there continued to be a suggestion on check-up roentgenograms that a malignancy might be present, it was determined to do an exploration of the left chest.



A. *B.*

Fig. 6, *A* and *B*.—(Case 8) Sharply circumscribed shadow in left upper lobe due to bronchiectatic cysts beyond bronchi blocked by mucoid impactions.

Operation: On June 26, 1950, the left chest was explored. There were fine avascular adhesions between the cystic area in the left upper lobe and the neighboring left lower lobe and chest wall. There were more vascular adhesions holding the apex of the left upper lobe to the dome of the chest. The cystic areas could be seen on the surface of the subapical division of the left upper lobe, and a hard area could be palpated in which there was mucoid impaction which ran down into the hilum of the lung. The apical and subapical segments of the left upper lobe were removed.

Pathologic Report: Specimen consisted of the upper two-thirds of the left upper lobe. In the bifurcation of the segmental bronchi of this segment of lung were two mucoid plugs. One plug measured 3.1 by 1.5 cm.; the second plug 3.6 by 2.3 by 1.2 cm. These plugs caused the bronchi at the point of bifurcation to become greatly dilated and to have very thin yellowish linings. Distal to the mucoid plugs was a multilocular cavity which measured approximately 4.4 by 3.5 by 2.3 cm. The mucoid plugs had a greenish-gray-white appearance and were puttylike in consistency. The cut surface revealed a thick, tenacious, ropy mucoid material.

The patient's convalescence was uneventful, but she is still subject to attacks of asthma.

Two patients have not had surgery. The history, roentgenogram findings, and course of their pulmonary disease seem to clearly indicate that they have had mucoid impaction of the bronchi.

CASE 9.—J. H., aged 18 years, was hospitalized in September, 1943, because of pneumonia in the right upper lobe. This cleared after a long convalescence, but left shadows in the right upper lung field. In September, 1946, she had an hemoptysis, and has had repeated hemoptyses since that time. There has been no recurrence of the cough and expectoration or fever that marked the initial illness. This patient had been asthmatic almost all of her life. Right upper lobe lobectomy was advised because of the repeated hemoptyses, but this was not accepted by the patient. The patient did return to the office, however, at intervals for check-up examinations. Small hemoptyses persisted, but these never became alarming. In view of the underlying history of asthma and the appearance of the roentgenograms, it was felt that a diagnosis of mucoid impaction of the bronchi was justified. Undoubtedly, there is a cystic bronchiectasis in the apical divisions of the right upper lobe. It may be necessary at a later date to do a resection because of the residual destruction of the lung.

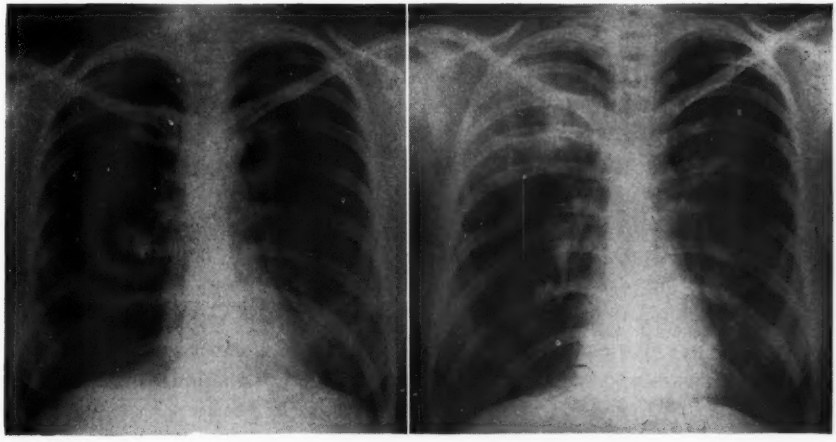
CASE 10.—Mrs. C. W., aged 29 years, had a history of allergy for ten years with asthma and hay fever. As a child she had been considered an asthmatic. In July, 1945, she had what was diagnosed as virus pneumonia. She coughed for several weeks. Penicillin therapy was used at that time both intramuscularly and by inhalation. In February, 1947, an asthmatic attack became severe and prolonged, and was not relieved by adrenalin. A bronchoscopy was done at that time. In November, 1947, she began to cough and has had a rather persistent cough ever since that time. She changed her climate from that of the Panhandle region of Texas to Tucson, Arizona. This did not improve her symptoms greatly, and during that time an abscess developed in her lung. A review of the films brought by the patient showed that she had had, in the past five years, infection in the right upper lobe with abscess, left lower lobe, and now left upper lobe (Fig. 7*A*, *B*, *C*, and *D*). Because of the widespread condition and the fact that a bronchogram showed cylindrical bronchiectasis in all lobes, surgery was not advised. It was considered that we were dealing with mucoid impaction of the bronchi with bronchiectasis and abscess formation.

DISCUSSION

This condition must be suspected when pulmonary disease occurs in asthmatics and individuals having chronic obstructive bronchitis. The presence of the mucoid impaction causes a febrile episode. The fever may be initially high but responds well to chemotherapy and then persists as a low-grade daily temperature elevation. The associated cough is not severe. It may be a dry cough or productive of small amounts of mucopurulent secretion. General lassitude and a slight localized pleuritic pain may be present. Hemoptyses occurred in three of ten patients in our series.

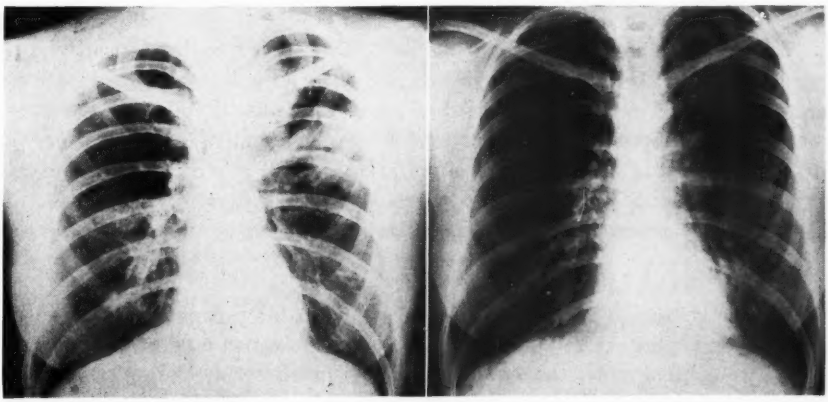
Adequate sputum studies should differentiate this condition from tuberculosis. Differentiation from a putrid abscess may be more difficult. The history of asthma, the milder clinical course, the nonputrid sputum, and the position of the abscess cavity within the lung usually will make this differentiation possible. The eosinophil count was elevated slightly to twelve and fourteen in two of the patients in this report, but in the others the count was in the normal range. It may be impossible to rule out new growth in the lung without doing a thoracotomy. The surgeon, in operating upon such patients, must be on guard and must establish the true diagnosis before becoming committed to a too radical resection of pulmonary tissue.

The diagnosis depends upon a careful history, laboratory studies, interpretation of roentgen films, and an awareness that the condition does exist in patients with an underlying asthmatic diathasis. Bronchoscopy is of little diagnostic aid because of the inaccessibility of the impaction. Occasionally, the diagnosis can be made with certainty when the patient gives a history of having coughed up a large, hard mucous plug.



A. *B.*

Fig. 7, *A* and *B*.—(Case 10) Soft rounded shadow in upper pole of right hilum. Two months later a large abscess has formed beyond the blocked bronchus. Note new hilar shadows in left lung.



C. *D.*

Fig. 7, *C* and *D*.—Three months later right upper lobe now clear. Dense shadows have appeared in left upper lobe. Eleven months later lung fields appeared clear.

NONSURGICAL MANAGEMENT

Where a definite diagnosis of mucoid impaction of the bronchus can be made, an attempt should be made to relieve the patient of this condition by nonsurgical measures. These include the use of chemotherapeutic agents, expectorant cough mixtures, and bronchodilators. Bronchoscopy is usually not helpful because of the inaccessability of the mucoid plugs. It has been suggested by some radiologists that roentgenogram therapy over a wide port in relatively small dosage acts as an expectorant and will aid these patients to rid themselves of the mucous plugs, thus improving bronchial drainage. We have had no experience with this type of therapy. ACTH also may be of benefit to these patients.

INDICATIONS FOR SURGERY

Indications for surgery in this condition are (1) persistence of suppuration with destruction of pulmonary tissue beyond impactions of mucus, (2) repeated hemoptyses from the associated cystic bronchiectasis, (3) the necessity of ruling out neoplasm when diagnosis is not certain.

This condition in its acute phase may appear to be life threatening, and yet can clear quickly leaving little demonstrable damage to pulmonary tissue. Due to the underlying asthmatic diathasis, these episodes may repeat in other segments of the lungs so that resection of pulmonary tissue must be limited to the actual lesion to preserve respiratory function. At the time of exploration the bronchial wall itself may have to be exposed to rule out neoplasm. With mucoid impaction the tumor is entirely within the bronchus. The bronchial wall is reddish brown in appearance and is edematous and partially necrotic. In certain instances, it will be necessary to resect an entire lobe rather than a segment so that the section through the bronchus can be made through normal tissue.

In certain individuals having this condition, surgery either is contraindicated or should be advised only because of compelling reasons. In a patient who has been followed over a period of years where multiple lesions have appeared and disappeared in segments of all lobes it would seem poor wisdom to attack a segmental atelectasis or suppuration beyond a plugged bronchus at any particular time. This patient has undoubtedly suffered grave damage to the pulmonary reserve and can ill afford to lose pulmonary tissue.

PROGNOSIS

The prognosis of this complication of asthma is the prognosis of the underlying disease. One can satisfactorily eradicate by resection a destroyed segment or lobe thus eliminating the symptoms of the complication. This does not insure that the patient will not repeat this same process in the future. In four of the ten patients that we have observed there have been recurrences of pneumonitis in other segments of the lung that were undoubtedly due to mucoid impaction of the bronchi.

SUMMARY

1. Mucoid impaction of the bronchi is a complication of asthma or chronic obstructive bronchitis.

2. The etiology and pathology of this clinical entity is discussed.

3. The roentgen appearance of the shadows cast by this disease may simulate tuberculosis, abscess and neoplasm.

4. Cystic bronchiectasis and fibroid pneumonitis are residual sequelae to this bronchial obstruction caused by mucoid impaction.

5. Surgery may be indicated to rule out neoplasm and/or to remove segments of lung destroyed by suppuration beyond the impaction.

6. Ten patients having this condition are reported. Eight had pulmonary resections. Two are being observed on nonsurgical management.

DISCUSSION

DR. LYMAN A. BREWER, III, Los Angeles.—The Association is indebted to Dr. Shaw and Dr. Paulson for bringing this condition to our attention in such a striking fashion. We have been interested in this problem for some time. The most important etiologic factors are: (1) an abnormal source of mucous secretion in the bronchial tree, and (2) conditions favoring retention of this mucus. Retained mucus may have an important effect on the spread and persistence of infection in other inflammatory conditions, particularly pulmonary tuberculosis. It will be well for us to consider various inflammatory diseases from this point of view.

I should like to comment briefly on only one phase of this problem, the persistence of mucous plugs in the bronchus as an important factor in producing pneumonitis and atelectasis in bronchiectasis. Here you have an abnormal source of mucus which, on occasion, is retained. In studying our resected pulmonary specimens, for bronchiectasis, we have found that lobar, segmental, and subsegmental atelectasis may be secondary to mucous blockage of the bronchus. In most instances this finding is not of great clinical importance; however, a most difficult problem is presented in the patients of the older age group who do not present histories typical of bronchiectasis and who have findings of bronchial obstruction that are impossible to differentiate from those of bronchiogenic carcinoma. The following case shows the difficulties that are encountered.

(Slide.) The patient was a 65-year-old waitress, complaining of severe hemoptysis of two weeks' duration. Roentgen examination of the chest revealed infiltration and pneumonitis with a possible central rarefaction in the lower right lung field. These findings pointed to an acute pulmonary abscess, possibly on the basis of bronchiogenic carcinoma. There was a history, on further questioning, that she had had a slight cough, productive of rather small amounts of mucoid sputum for a number of years, but no previous severe pneumonia. There was no evidence, as in the cases of Dr. Shaw and Dr. Paulson, of definite asthmatic bronchitis or bronchial asthma. She was put on intensive antibiotic therapy.

(Slide.) Two weeks later the roentgenogram showed a slight clearing of the lesion in the right lung field. The lateral film depicts atelectasis of the middle lobe. Antibiotics were continued, and she was symptomatically improved.

(Slide.) Bronchoscopy revealed no obstruction of the orifice of the right middle lobe. Purulent exudate issued from the right middle and lower lobe orifices. The roentgenogram demonstrated an enlargement of the shadow occupying the right middle lobe, similar to the cases presented by the speaker. Exploratory thoracotomy was advised. However, it was not until she brought up some foul sputum that consent to operation was obtained.

(Slide.) At exploration, the middle lobe was enlarged, boggy, and consolidated. Following removal of this lobe, cut section demonstrated a mucous plug which occurred exactly at the bifurcation of the two segments of the middle lobe, and thus could not be seen on bronchoscopy. On examination of the mucous plug, we could not see the laminations that the authors describe. It was degenerated mucus, with a good many fibrin strands, and

bacteria were present which were apparently saprophitic. The chronic pneumonitis, suppuration, and lung abscess formation were present beyond the bronchial obstruction. In this case the bronchiectasis was the source of the sputum which became inspissated and obstructed the segmental branches of the bronchi. Secondary pneumonitis and abscess formation resulted. In patients in the cancer age group, it is impossible to rule out bronchiogenic carcinoma and, contrary to the cases in younger persons presented by the authors, surgical exploration is very definitely indicated.

DR. JEROME HEAD, Chicago.—This seems to me an original and excellent paper. I have never knowingly encountered this entity, but I am sure this has been due to a failure in observation. The subject interests me greatly because I am greatly interested in the sequellae of segmental and subsegmental bronchi. I would like to bring up the question as to why this entity does not occur more often. We know that plugs of mucus can and frequently do plug main bronchi, in which case there is atelectasis of a lobe. If plugs of mucus obstruct main bronchi occasionally it is reasonable to believe they obstruct segmental bronchi more frequently and finer bronchi very frequently.

In spite of this it is quite obvious that we do not encounter segmental atelectasis or subsegmental atelectasis. This can be explained only on the basis that there is a collateral air cirulation between lung segments and lung lobules. If it were not for this collateral circulation it seems certain that every bronchitis, by plugging the finer bronchi, would become a suffocating bronchitis. Without this collateral circulation there would develop a spreading patchy atelectasis from obstruction of small bronchioles. Because no air would get in behind these plugs to blast them out, the patient would eventually suffocate from a progressive patchy atelectasis. This of course does not happen. If it were not for collateral circulation, lobular atelectasis would be infinitely more frequent than lobar atelectasis and would be as difficult to relieve. Lobar atelectasis of course is relieved only when one interferes actively to get rid of the plug of mucus. This active intervention is necessary because no air can get in behind the plug to blast it free. I believe, therefore, that we should not speak of segmental or lobular or patchy atelectasis. When the parenchyma distal to an obstructed segment or lobule becomes opaque it becomes opaque because of infection and not primarily because of airlessness. To go further, we must ask ourselves what happens to an obstructed segment or lobule when infection does not occur. I believe we will find in these lung areas the development of the opposite of an atelectasis, in other words an emphysema. This is based on the assumption that the air enters the segment through the collateral circulation more easily than it passes back through the same channels. In this case the effect of continued coughing is to extend and stretch the obstructed segment. That this does occur is suggested by the following observations:

At the present time we have under observation a man in whom a single segment of the upper lobe which has been obstructed by a calcified gland has dilated to occupy the upper half of the thorax crowding the rest of the upper lobe and the lower lobe into the lower thorax. In another case in which there appeared to be a bleb occupying the lower half of the thorax operation revealed a tremendously emphysematous accessory lobe. This was connected to the lower lobe by a pedicle of lung tissue. There was no direct bronchial communication. In this case the air had passed into the accessory lobe through collateral alveolar channels. Apparently it was entrapped there and the result was a vesicular emphysema.

If we bear in mind the possibility that obstruction of a segmental or subsegmental bronchus without infection causes emphysema rather than atelectasis I believe that we will be on the way to recognizing an important clinical entity.

DR. RICHARD H. OVERHOLT, Brookline, Mass.—This is an extremely important subject, particularly from the point of view of differential diagnosis in carcinoma, inflammatory lesions, cysts, and bronchostenosis. Also, in some cases of severe asthma a mucous plug may act as a trigger mechanism to set off bronchospasm. If such a condition can be found and eliminated by excision further attacks of asthma may disappear.

It was not quite clear to me, from what has been said, whether or not the mucous plug in the reported cases was the primary or secondary factor. In instances of this kind that we

have observed there has been some condition which caused bronchial occlusion. The plug seemed to have resulted from concentration of mucus distal to the point of obstruction, therefore making it a consequence of another factor.

I should like to ask Dr. Shaw if they were not able to find in the pathologic specimens evidence of pressure from a gland, stenosis, or other organic change that might explain why the mucous plug developed.

DR. DEAN B. COLE, Richmond, Va.—Dr. Shaw has presented the problem of mucoid impaction beautifully but, as he has indicated, it is not new. The treatment of this condition rarely, if ever, is surgical. We have done lung mappings on many asthmatics in whom we have encountered bronchial spasm and mucoid impaction of bronchi. We have been able to demonstrate a reversible bronchiectasis in one such instance.

We have seen Loeffler's syndrome a number of times, and occasionally this will move from one lobe to another until eventually all lobes have been involved.

DR. SHAW (closing).—I wish to thank Dr. Brewer, Dr. Head, Dr. Overholt, and Dr. Cole for their discussion. In answer to Dr. Overholt's question I cannot be certain what is the actual underlying cause. I think the major cause is the thick tenacious type of mucus that these particular individuals have. In other words, it is similar to the child with chronic pancreatitis associated with bronchiectasis. Why the first accumulation occurs I do not know. Probably some thick material gathers in one bronchus, and it is my feeling that the accumulation from one bronchus, causing pressure obstruction on the neighboring branch bronchus, causes the formation of these plugs which is rather typical of this condition. In none of our patients have we found any significant glandular obstruction, but of course we are seeing them after suppuration has taken place, late in the process rather than when it began.

I did not mean to suggest, as Dr. Cole pointed out, that this is anything new. I did search the literature for a period of ten years and could find no direct reference to this particular entity. There are many reports describing atelectasis in asthma and Loeffler's syndrome. So far as I can tell no one has actually described the pathology of Loeffler's syndrome. They merely described the patchy, evanescent shadows that appear in the roentgenograms of people having this condition. It is my main concern to differentiate mucoid impaction from other diseases which have an entirely different prognosis. I feel that in some of these cases we will be forced to operate in order to rule out a disease that has a more severe prognosis and requires another form of treatment.

I also feel that in one of our cases, as Dr. Cole described, and as shown in the main body of the paper, we have had shadows of the same sort appear in every lobe of the lung, and the bronchogram demonstrates bronchiectasis in every lobe. This patient is not, however, in a good respiratory state as Dr. Cole suggests. She does have definite dyspnea, and I think she has suffered irreparable damage to the pulmonary bed. I certainly would not suggest that anyone should remove a segment of her lung at any particular time impaction should occur and a segmental atelectasis be found.

THE TREATMENT OF CARDIOSPASM: ANALYSIS OF A TWELVE-YEAR EXPERIENCE

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THE experience of the Mayo Clinic in the management of cardiospasm has been the subject of several previous reports from this institution. In 1906, Henry S. Plummer[1] recorded the first eleven cases of cardiospasm treated by him at the clinic, and he described a technique of dilatation which with relatively little modification has been employed in the great majority of approximately 1,800 patients with cardiospasm treated endoscopically at the clinic. In subsequent reports, Plummer[2, 3] reviewed his ever-increasing experience in the management of cardiospasm and his methods were either adopted or modified by other authorities interested in the treatment of this condition. In 1921, Plummer and Vinson[4] reported 301 cases, and the accumulated experience has since been described, in subsequent articles, by Vinson[5] and by Moersch.[6-8] Various problems in the diagnosis and management of cardiospasm have been the subjects of communications by other members of the staff[9-11]; however, there has been no complete review of the accumulated experience in the management of the cases of cardiospasm at the clinic since the report of Moersch[8] in 1936.

It is the principal purpose of this paper to describe and evaluate the results of treatment in 601 consecutive cases of cardiospasm in which dilatation was employed in the twelve years between Jan. 1, 1935, and Jan. 1, 1947. In addition, our experience with all cases of cardiospasm treated surgically at the clinic is reviewed. The etiologic and diagnostic aspects of cardiospasm will be reported in more detail in other communications.

DEFINITION OF CARDIOSPASM

Cardiospasm is a nonorganic stenosis of the lowermost portion of the esophagus. The portion involved by this closure is at, and immediately above, the juncture of the esophagus and stomach and is seldom more than 4 to 6 cm. long. Synonyms for cardiospasm include achalasia, mega-esophagus, idiopathic or diffuse dilatation of the esophagus and preventriculosis. All refer to obstruction at the site of the physiologic sphincter at the cardia (Fig. 1).

In order to evaluate the treatment of cardiospasm, it is most important that it be differentiated from other forms of esophageal spasm. The roentgenologic and clinical characteristics of diffuse spasm of the esophagus were described by Moersch and Camp[12] in 1934, and further elaborated upon by Schmidt[13] in 1939. This type of spastic constriction involves the smooth muscle portion of the esophageal musculature and may affect the lower third to two-thirds of the esophagus. The roentgenographic appearance of diffuse spasm

Read at the Thirty-first Annual Meeting of the American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

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varies considerably (Fig. 2). Hence, diffuse spasm of the esophagus is often referred to as pseudodiverticulosis,[14] localized spasm, segmental spasm, nonsphincteric spasm, reflex spasm, or even esophagitis. Many authorities have implied that diffuse spasm is an early stage of cardiospasm. In our experience, diffuse spasm is a clear-cut clinical entity not to be confused with cardiospasm, even though they may coexist occasionally. At times there may be some difficulty in distinguishing these conditions, both clinically and roentgenologically. However, the two entities may be readily differentiated by the passage of an olive-tipped bougie (41 F.) over a previously swallowed thread. In cardiospasm the obstruction is localized to a short segment of esophagus at the cardia. In diffuse spasm obstruction begins in the midesophagus and extends all the way to the stomach. The alteration in the roentgenoscopic appearance of the esopha-

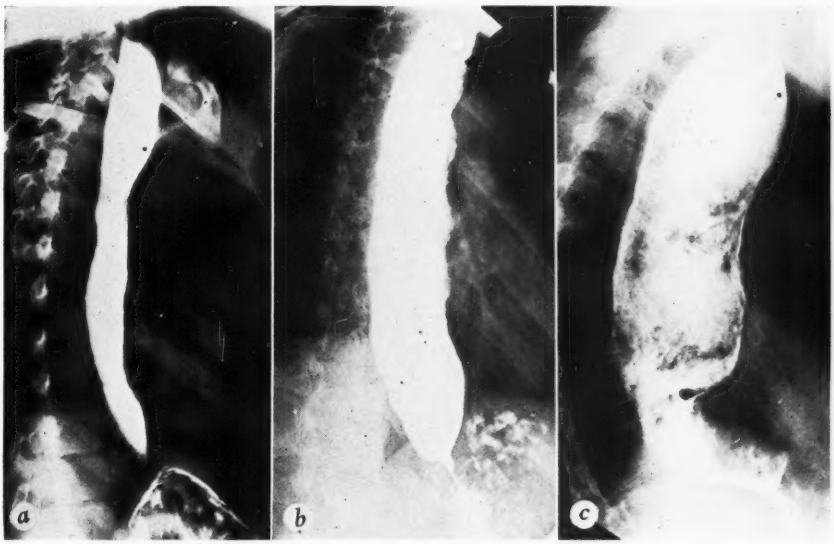

Fig. 1.—Cardiospasm. *a*, Moderate dilatation; *b*, diffuse dilatation; *c*, marked dilatation, angulation, and elongation.

gus following dilatation of the cardia in cardiospasm should be mentioned. The activity of the esophageal musculature often is increased and the roentgenoscopic appearance may closely simulate that of diffuse spasm. This fact must be taken into consideration by the roentgenologist in his evaluation of conditions after treatment (Fig. 3).

Particular effort must be made to distinguish cardiospasm from other types of esophageal spasm. Diffuse spasm of the esophagus does not respond to treatment in the same manner as cardiospasm, and the hydrostatic dilator should not be used in cases of nonsphincteric spasm. Esophageal spasm which may be associated with other lesions of the esophagus or gastrointestinal tract, such as esophageal diverticula, hiatal hernia, peptic ulcer of the stomach or duodenum, and disease of the gallbladder also must be differentiated from cardiospasm.

TECHNIQUE OF DILATATION

Russel[15] described the first pneumatic dilator in 1898. Actually, Plummer's hydrostatic dilator was a modification of the Russel dilator adapted for water pressure and passed over a previously swallowed thread. The pneumatic principle was combined with Hurst's mercury-weighted tube by Tucker[16] and also by Browne and McHardy.[17] Some authorities continue to use dilators which are introduced through the esophagoscope.[18, 19]

The methods used at the clinic for dilating the cardia of patients with cardiospasm were described by Moersch[7] in 1933 and are essentially unchanged today. However, it is desirable to outline again the technique and procedure that we employ.

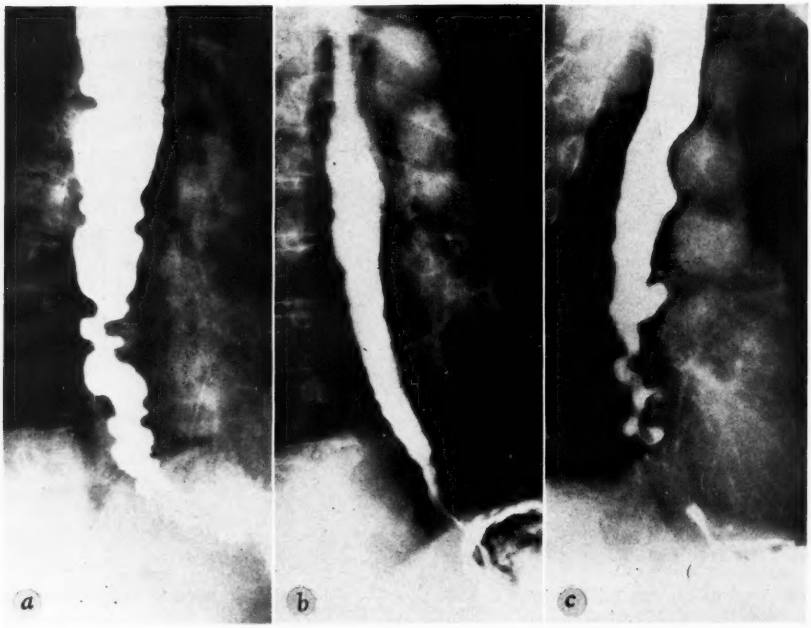

Fig. 2.—Diffuse spasm of esophagus. *a*, Diffuse irregular spasm; *b*, diffuse constant narrowing; *c*, pseudodiverticulosis.

Dilatations are carried out over a previously swallowed twisted silk thread. At present we are supplying our patients with "EE" buttonhole silk. The patient starts to swallow his thread twenty-four hours prior to the first dilatation; he swallows the thread slowly at the approximate rate of one foot (30.5 cm.) every hour and continues until fifteen feet (467 cm.) have been swallowed. He finds that eating food and swallowing liquids help him get the thread down, and he is permitted to eat all meals except the one prior to his treatment. During the night the thread hangs from the mouth, but it is usually advisable for him to place it between two teeth in order to avoid chewing it through during sleep. Treatment is not begun until the thread is completely anchored. Oc-

casionally it is necessary to weight the end of the thread with lead shot. Nearly all patients are successful in getting a thread in place if they persist in their efforts, no matter how marked the obstruction.

Once the thread is firmly anchored, preliminary dilatations with metal sounds are carried out. The patient sits in a low chair facing the physician. A 41 F. olive-tipped metal bougie is attached to the end of a flexible whalebone staff, and this is passed over the thread into the stomach (Fig. 4, *a*). A second dilatation follows immediately, using a 50 or 60 F. Plummer sound guided over the thread by a flexible wire spiral (Fig. 4, *b*). Following the passage of these metal sounds, the patient rests for forty-eight to seventy-two hours. He is

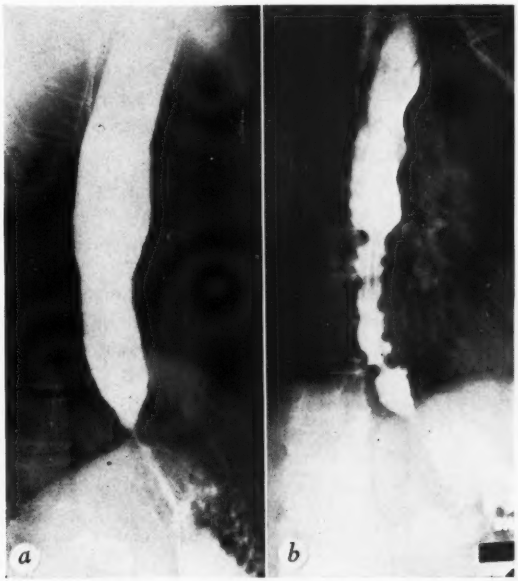

Fig. 3.—Effect of treatment on roentgenographic appearance of cardiospasm. *a*, Diffuse dilatation of esophagus before therapy; *b*, irregular and segmental spasm after hydrostatic dilatation.

usually relieved of the greater part of his dysphagia and gets considerable food through the cardia during this period. The preliminary dilatation with sounds is important from a diagnostic standpoint, as the nature of the obstructing lesion can usually be determined by the "feel" of the sound as it passes through the cardia. Furthermore, subsequent hydrostatic dilatation is easier because esophageal retention has been relieved. Esophagitis improves rapidly following the initial dilatation and, in our opinion, the preliminary passage of sounds reduces the risk of the hydrostatic dilatation.

When esophagoscopy is necessary for dilatation, the stenotic cardia must be located and a small flexible woven bougie, a stomach tube, or a small sound may be passed into the stomach. Then the esophagoscope itself may be passed into the stomach or a thread may be tied to the distal end of a bougie tube or sound, the esophagoscope withdrawn, and dilatations carried out over the thread.

The hydrostatic dilator has been described elsewhere.[7, 20] A flexible, whalebone staff is passed through the rubber tube, and a 41 F. olive-tipped metal bougie is attached to the threaded end of the whalebone staff (Fig. 4, *c*). The dilator is connected to the water source and pressure gauge and is tested before every treatment. This dilator is passed over the previously swallowed thread to insure the safety of the procedure, also to insure the proper placement of the bag in the cardia. The success of the procedure depends on exact placement of the bag before it is inflated. Just before passing the hydrostatic dilator,

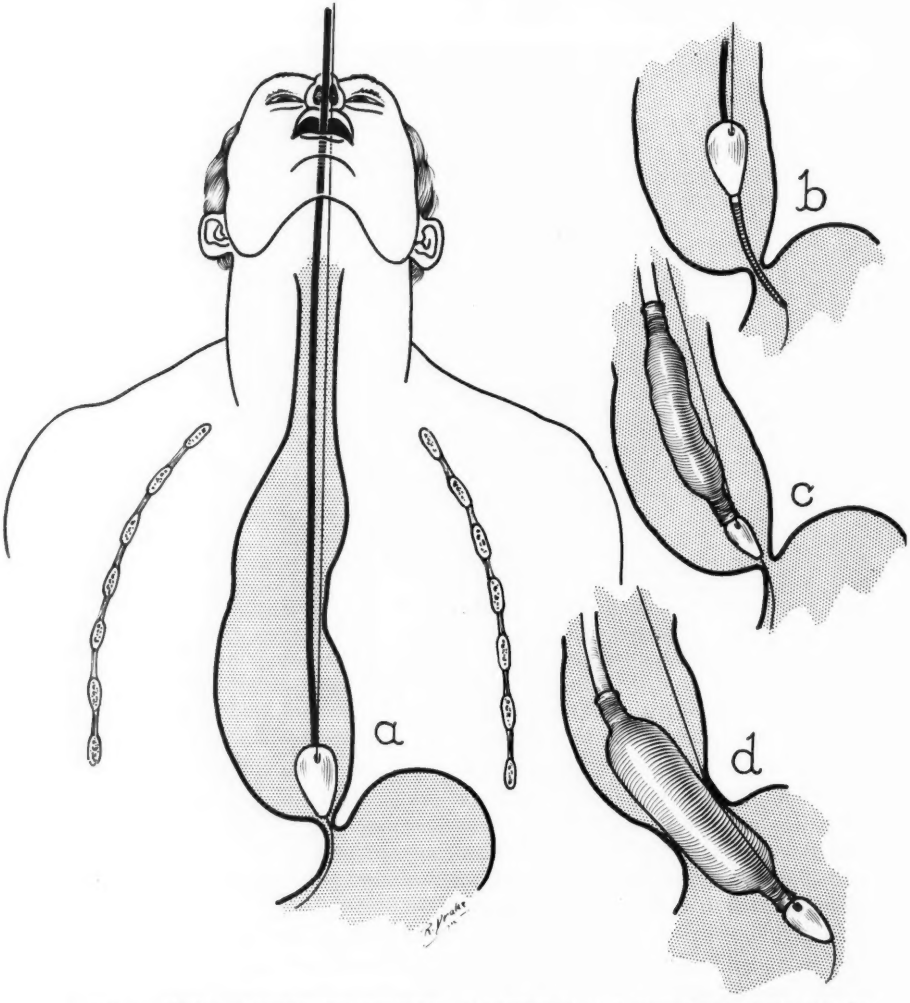

Fig. 4.—Method of performing dilatations. *a*, Passage of 41 F. olive-tipped bougie into the stomach; *b*, a 50 or 60 F. sound is passed in o the stomach guided by a flexible wire spiral; *c*, the hydrostatic dilator is passed to the cardia; *d*, distention of the hydrostatic dilator across the cardia.

a 41 F. olive is passed into the stomach (Fig. 4, *a*). The exact distance from the point of the obstruction to the upper incisor teeth is determined and is marked off on the tube of the hydrostatic dilator. Thus the physician knows just how far to pass the hydrostatic bag. Once in place the bag is carefully inflated (Fig. 4, *d*) to 22 to 24 feet (9.5 to 10.3 pounds per square inch) of water pressure and the pressure maintained for several seconds. The patient usually will have considerable pain at the time of dilatation if the bag is properly placed. This pain subsides rapidly after the dilator is withdrawn. One evidence of a successful dilatation is the presence of a small amount of fresh blood on the distal end of the dilator.

The beneficial effects of the dilatation are immediate, and the patient spends the next two or three days testing his ability to swallow. He is advised to eat meat, apples, popcorn, and any other foods which may have given him particular difficulty. If complete relief from dysphagia is not obtained, the patient again swallows a thread, and a second hydrostatic dilatation is performed forty-eight or seventy-two hours after the first. It is seldom necessary to perform more than two or three hydrostatic dilatations, and the entire treatment program is usually completed in a week or ten days.

CASES STUDIED

We selected the period from Jan. 1, 1935, through Dec. 31, 1946, for our review of results of treatment of cardiospasm by hydrostatic dilatation for several reasons. Patients seen prior to Jan. 1, 1932, were covered in a previous report, and it was difficult to trace patients treated prior to 1935. We terminated our study at the end of 1946, so that the minimal period of follow-up would be four years. Some of the patients, therefore, have been under observation for sixteen years. Results based on a study of 601 patients, more than 75 per cent of whom have been successfully traced, should be statistically significant.

Not included in our series are sixty patients who were treated for cardiospasm during the twelve years, but who had also undergone dilatation at the clinic before 1935. Thus all our patients were new patients in the sense that we had not treated them for cardiospasm before. Of the 601 patients, 205 had had some type of dilatation therapy before we saw them.

For thirty-nine patients it was necessary to pass an esophagoscope to aid in the dilation. Forty-six patients were treated by passage of sounds without hydrostatic dilatations. The hydrostatic dilator was not used on some patients because of extreme youth, old age, or some associated serious disease. A few of these patients were so gratified by the temporary relief afforded by bouginage that they did not remain for hydrostatic dilatation. Four children, 6 years of age or less, had treatment by sounds only, and three obtained poor results. The fourth child has not been traced. Ten of the 46 patients (22 per cent) received satisfactory benefits from the passage of sounds alone. Vinson[20-21] and Carr[22] stated that only 10 per cent of patients treated with bougies alone received permanent relief.

In the period under consideration we treated 555 new patients with the hydrostatic dilator. For 452 of these patients, adequate follow-up studies were

available and our conclusions concerning the method of hydrostatic dilatation are based on a study of these 452 patients.

RESULTS OF TREATMENT BY HYDROSTATIC DILATATION

We have set rather exacting criteria for satisfactory treatment. A good result from dilatation implies that lasting relief from dysphagia follows a course of treatment. In our study, results were classified as satisfactory or unsatisfactory. A satisfactory result means that the patient has had no dysphagia or only occasional or transient difficulty with swallowing since treatment. Patients in the satisfactory classification have remained virtually asymptomatic for four years or more* after treatment. A recurrence known to have developed in less than four years after treatment was classified as an unsatisfactory result. Also included as having unsatisfactory results were those individuals who reported partial relief of dysphagia but who still had significant symptoms (Table I).

We shall consider our experience in three categories: (1) results of the first course of treatment, (2) results of a second course of treatment for those patients who derived temporary relief of dysphagia after the initial course and who returned for further treatment, and (3) results of further treatment for patients who made three or more visits because of recurring dysphagia.

The Initial or First Course of Treatment.—Following preliminary passage of metal sounds, all of the 452 patients had one or more hydrostatic dilatations. The average number of hydrostatic dilatations was 1.67 per patient. The highest number for any individual patient at the initial visit was 12.

Of the 452 patients, 272 (60.2 per cent) were relieved of dysphagia for periods of four to sixteen years and are considered as having obtained satisfactory results. In the remaining 180, dysphagia recurred in less than four years, and the results were unsatisfactory according to our criteria.

Second Course of Treatment.—Ninety-four of the 180 patients who obtained an unsatisfactory result from the first course returned for a second course of treatment, and further hydrostatic dilatations were administered. Thirty-six of these 94 (38.3 per cent) obtained relief of dysphagia for four years or more and the results are classified as satisfactory. In fifty-eight cases, however, recurrence developed, and the results are considered unsatisfactory.

Three or More Courses of Treatment.—Twenty-six of the fifty-eight patients who obtained unsatisfactory results from a second course of treatment returned one or more times after the second course for further hydrostatic dilatations. Only five (19.2 per cent) of them were satisfactorily relieved by additional treatment.

TABLE I. RESULTS OF TREATMENT BY HYDROSTATIC DILATATION OF 452 TRACED PATIENTS

COURSE	PATIENTS TRACED	SATISFACTORY RESULTS		UNSATISFACTORY RESULTS	
		NO. OF PATIENTS	PER CENT	NO. OF PATIENTS	PER CENT
1	452	272	60.2	180	39.8
2	94	36	38.3	58	61.7
3 or more	26	5	19.2	21	80.8

*A few patients known to be asymptomatic for more than two years but lost to follow-up thereafter are included in the satisfactory grouping.

All Courses.—It is evident that patients who did not respond satisfactorily to the initial course of treatment were less likely to obtain good results following a second course (38.3 per cent) or from any number of additional courses of treatment (19.1 per cent). Hence, if hydrostatic dilatation does not produce lasting relief of dysphagia at the first or second course, the prospects for success from further dilatation are not bright.

If the total number of satisfactory results in Table I are added, we find that 313 (69.2 per cent) of our 452 traced patients obtained satisfactory and lasting results; however, we did not have the opportunity of treating a second time all of the patients who had unsatisfactory results after the first course, nor did we treat all of those who failed to obtain satisfactory results from a second course. Had we been able to treat all of these patients and if the same percentage of satisfactory results noted in Table I had been obtained, our total number of satisfactory results would have been 341 (75.4 per cent) for two courses of treatment, or 362 (80.1 per cent) for all courses. Hence, if given the opportunity, we could anticipate satisfactory results from hydrostatic dilatation in 80.1 per cent of cases.

Number of Dilatations.—An analysis of the number of dilatations required in each case was made. When only one or two hydrostatic dilatations were necessary at the initial visit, satisfactory results were obtained in 64 per cent of the cases. However, if three or more dilatations were required to relieve dysphagia, the results were satisfactory in only 38 per cent. The experience was similar when patients returned for subsequent courses of treatment. This is further evidence to indicate that a favorable result can be anticipated from a prompt response to dilatation. If multiple treatments are required, the outlook is not as good.

In a large number of patients with cardiospasm there are many variable factors that must be considered in an evaluation of therapy. We have made an attempt to correlate some of these factors with the success of treatment.

Relation to Age.—Patients of all ages were treated, and, except for the very young, results were comparable in all age groups.

Fourteen patients treated by hydrostatic dilatation in this group were 15 years of age or less. Ten of these children were 11 to 15 years of age. Seven of the eight children in this age group who were traced did well. Results thus were comparable to those for adults. However, for the four children who were less than 10 years of age, results were not good. Only one got a good or satisfactory result. Results of use of sounds in young children were mentioned previously.

Relation to Duration of Symptoms.—The relationship of the duration of symptoms to the success of treatment is indicated in Table II. The ratio of

TABLE II. CORRELATION BETWEEN DURATION OF SYMPTOMS AND SUCCESS OF THERAPY OF 452 TRACED PATIENTS

DURATION OF SYMPTOMS (YEARS)	PATIENTS TRACED	SATISFACTORY RESULTS	
		PATIENTS	PER CENT
0-1	78	53	68.0
1-5	212	150	70.8
6-10	83	58	69.9
More than 10	79	52	65.8

satisfactory to unsatisfactory results is remarkably constant, regardless of the duration of the disease.

Relation to Size and Shape of the Esophagus.—The relationship between the size of the esophagus and the result of treatment is shown in Table III. Results were somewhat less favorable in the slightly enlarged and markedly enlarged organs (Fig. 1, *c*) than in the moderately and diffusely enlarged organs (Fig. 1, *a* and *b*). It is interesting that the slightly and markedly dilated esophagi responded less well to surgical treatment. It should be pointed out that in some cases of cardiospasm in which marked enlargement of the esophagus was present dilatation was impossible, and such cases have not been included here. In the surgical group there were eight such cases. Had these eight patients been taken into consideration in Table III, our results in the cases of marked enlargement of esophagus doubtless would have been less satisfactory than those presented. Our percentage of satisfactory results would have been about 58 per cent.

TABLE III. CORRELATION BETWEEN SIZE OF ESOPHAGUS AND SUCCESS OF TREATMENT OF 452 TRACED PATIENTS

DEGREE OF ENLARGEMENT OF ESOPHAGUS	TRACED PATIENTS	SATISFACTORY RESULTS	
		NUMBER	PER CENT
Slight	55	31	56.4
Moderate	200	139	69.5
Diffuse	127	98	77.2
Marked with torsion and angulation	70	45	64.3

A reduction in size of the esophagus may be anticipated in some cases after hydrostatic dilatation, although this diminution in size is uncommon in the markedly dilated esophagus. Actually, it is hard to predict with any certainty the likelihood of a decrease in size after treatment.

Relation to Type and Severity of Symptoms.—Obviously, results of treatment by dilatation were most dramatic in those patients who had marked obstruction and who regurgitated food most readily.

The relationship of pain to cardiospasm will be considered in another communication. In sharp contrast to figures given by some authors, pain occurred in less than 30 per cent of our cases. When pain was a prominent symptom of cardiospasm, we frequently obtained a less satisfactory result. Of 132 patients who complained of pain, only 51.5 per cent obtained satisfactory results, as opposed to 69.2 per cent for the whole group. In a number of cases our treatment was effective in relieving the obstruction and regurgitation of cardiospasm, but the patient continued to have pain.

Pain is a much commoner symptom in the syndrome of diffuse spasm of the esophagus than it is in cardiospasm. Although pain may be a feature of cardiospasm, an unusual amount of pain should make one wonder whether some other esophageal disease is associated.

Relation to Associated Disease.—Certain associated disease processes had an influence on the result of therapy. Patients who had associated pulsion diverticula of the upper or lower part of the esophagus were more difficult to

treat, and results were poor in this group. Patients who had hiatal hernia in association with cardiospasm were likewise less favorable candidates for treatment. We have learned that hiatal hernias of the short esophagus type are particularly troublesome and that dilatation of cardiospasm in such cases may be hazardous. The development of a short esophagus after rupture of the lower part of the esophagus has been noted. Fig. 5, *b* shows the condition after rupture of the lower part of the esophagus by the hydrostatic dilator. In retrospect there may have been a small hiatal hernia before dilatation (Fig. 5, *a*).

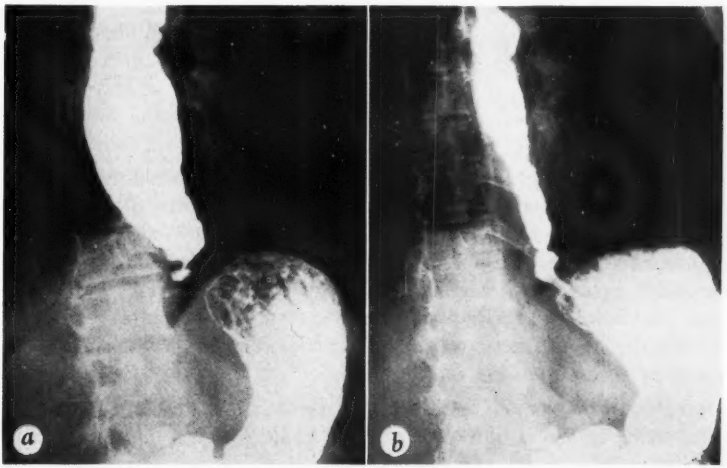

Fig. 5.—Development of short esophagus following rupture of lower part of esophagus by hydrostatic dilator. *a*, Cardiospasm before treatment; *b*, cardiospasm with small hiatal hernia.

In forty of our 601 cases, diffuse spasm of the esophagus was associated with cardiospasm. The diagnosis of the combined conditions can be suspected roentgenographically and confirmed by the findings at dilatation. When both conditions are present, results of treatment are difficult to evaluate. The obstruction of cardiospasm will often be relieved while the pain of diffuse spasm persists. The presence of associated diffuse spasm probably accounts for the less favorable results noted (Table III) in those patients with slight enlargement of the esophagus. The same considerations apply to surgical results (as will be evident in Case 3).

Painful Reactions.—As previously stated, most patients experience pain in the subxyphoid area during hydrostatic dilatation. This usually subsides rapidly after removal of the dilator. In twenty-one of our cases rather severe epigastric or substernal pain developed several hours after hydrostatic dilatation. This pain was often referred to the neck, ears, shoulders, or arms. None of these patients showed any objective evidence of esophageal perforation, nor were there any physical findings of significance. Some of them did have some fever, and several were kept in the hospital for observation. In none of these

cases were there any serious consequences. In our opinion, these painful reactions were the result of spasm of the esophagus secondary to the trauma of the procedure. Results of therapy in this group were comparable to those of the entire series.

COMPLICATIONS OF HYDROSTATIC DILATATION

The forceful dilatation of the cardia to a diameter of 3 to 4 cm. by a set of silk and rubber bags distended with water carries a certain risk. It is inevitable that a number of patients will experience undesirable symptoms or complications as the result of the treatment (Table IV).

TABLE IV. COMPLICATIONS FOLLOWING HYDROSTATIC DILATATION OF 555 CASES

TYPE OF COMPLICATION	CASES	MORTALITY
Esophageal "split"	10	2 (0.36 per cent)
Aspiration pneumonitis	6	0
Hematemesis	7	0

Hemorrhage.—Only seven patients had significant bleeding after hydrostatic dilatation. A small amount of blood on the hydrostatic dilator is expected after such dilatations, but serious bleeding is unusual. When hematemesis or melena occurs after dilatation, some associated process should be looked for, such as gastric ulcer, hiatal hernia, or associated stricture. Two of our patients who bled are known to have had short esophagus, and one patient had a gastric ulcer. One of these patients subsequently underwent esophagogastrostomy elsewhere with good results.

Aspiration Pneumonitis.—Aspiration pneumonitis developed after hydrostatic dilatation in six of our cases. All of these patients promptly recovered. It is our feeling that preliminary passage of sounds relieves esophageal retention and reduces the frequency of aspiration of esophageal content.

Rupture of the Esophageal Wall Near the Cardia.—This complication occurred in ten cases after hydrostatic dilatation. In such cases severe epigastric and substernal pain develops a few hours after dilatation. At the onset the pain is hard to distinguish from that of a painful reaction. However, almost invariably left pleuritic pain with both the physical signs and roentgenologic evidence of pleuritis appears. Effusion may develop and require thoracentesis.

Of these ten patients, two died of mediastinitis, one in 1937, and the other in 1940. In one case (1943), left empyema developed which required surgical drainage. This patient recovered but subsequently had a short esophagus type of hiatal hernia with stricture at the esophagogastric junction (Fig. 5, *a* and *b*). In another case a left transthoracic incision was made after the rupture, and the lower part of the esophagus and upper part of the stomach were resected. This patient did well, and is considered in the discussion of surgical management of cardiospasm. The remaining six patients made complete recoveries with conservative therapy.

The management of esophageal rupture was reviewed by Seybold, Johnson, and Leary.[23] When mediastinitis follows dilatation of the cardia, drainage of the posterior mediastinum is the safest procedure. When prompt surgical drainage is combined with adequate antibiotic therapy, the mortality from

hydrostatic dilatation should be negligible. Our two fatalities occurred prior to the era of penicillin and other effective antibiotics and before radical surgical treatment was considered feasible.

COMMENT ON TREATMENT BY HYDROSTATIC DILATATION

It is often stated that successful management of cardiospasm by the method of dilatation requires that treatments be administered at frequent intervals over long or indefinite periods of time. In our experience, this has not been the case.

The majority of patients who have cardiospasm can be successfully treated by hydrostatic dilatation. Not only are the immediate results satisfactory, but these patients have been relieved for periods ranging from four to sixteen years by one or two courses of treatment. It is also obvious that relief of dysphagia is usually accomplished by one or two hydrostatic dilatations. If permanent relief is not obtained during the first or second course of treatment, further dilatations are less likely to produce a favorable and lasting result.

Our studies show that approximately 20 per cent of patients cannot be expected to obtain permanent benefit by hydrostatic dilatations. A good many of these patients are improved but still have dysphagia; others have to return at intervals for repeated dilatations in order to control their symptoms. Although we do not consider the results satisfactory in these patients, the periodic dilatations enable them to live comfortable lives.

On the whole, the results of treatment in the present series of patients are comparable to those reported by Moersch.[7, 8] The experience of Nagel,[24] using methods similar to ours, is also comparable. In a group of fifty-seven patients, he obtained good results from dilatation in 73 per cent. Nagel also emphasized the point that a single forceful dilatation produces much better results than repeated "half-hearted attempts." Freeman[25] also favored the use of the hydrostatic dilator and apparently has had good results with one dilatation.

Successful dilatation therapy after the proper diagnosis of cardiospasm depends on two factors: (1) accurate placement of the dilator in the cardia, and (2) severe forceful dilatation of the physiologic sphincter by an expanding dilator. Obviously, the passage of simple bougies or mercury-weighted tubes does not often produce sufficient expansile force at the cardia to have any lasting effect. In most cases such procedures must be repeated frequently. In our opinion, the previously swallowed thread permits accurate placement of the dilator and greatly adds to the safety of the procedure. In each case the dilatation, however forceful, must be administered at the level of the constriction if favorable results are to be anticipated.

In a recent communication, Sifers and Crile[26] reported that only eleven of ninety-two patients treated by means of dilatation obtained satisfactory results; sixteen of their patients were considered improved, forty slightly improved, and eighteen unimproved. Their dilatations were performed with Hurst mercury bougies and the Browne McHardy pneumatic dilators. We must conclude that effective dilatation was not obtained in most of their cases. Similarly, Fritz, Clark, and Adams[27] advocated surgical treatment because of the "temporary results usually obtained by mechanical dilatation from above." Their temporary results do not conform to our experience.

We realize that the method of hydrostatic dilatation employed by us is a drastic procedure. There is nothing "half hearted" about it, and the procedure carries risk. Ochsner, DeBakey, and DeCamp[28] recently called attention to the 2.8 per cent mortality in the 804 cases reported by Moersch.[7] As previously stated, this was reduced to 0.36 per cent in the twelve-year period under consideration, and in the past eleven years there have been no deaths. We must anticipate a 3 per cent incidence of complications. However, with chemotherapy and prompt surgical intervention, mortality in the future should be negligible.

SURGICAL CONSIDERATIONS IN THE TREATMENT OF CARDIOSPASM

We believe that operative treatment should be considered in all cases in which hydrostatic dilatations or more conservative measures do not give satisfactory results. From this study it appears that about 20 per cent of patients will not respond to conservative methods of treatment such as dilatation. It is difficult to anticipate which patients will fail to respond to conservative measures, but in general those who have marked enlargement of the esophagus and retention are most likely not to respond.

Several of the operative procedures such as vagotomy, sympathectomy, and phrenicectomy are based on the neurologic concept of the etiology of cardiospasm. These operative procedures have given some satisfactory results but none have given uniform and consistently good results. One of the earlier surgical procedures was that advocated by von Mikulicz[29] of transgastric retrograde dilatation. This procedure gave only temporary relief and is now rarely, if ever, utilized.

The more common types of operative procedure utilized at present are directed to either short circuiting the obstruction or attacking the constricted area (Fig. 6). These procedures are as follows: (1) esophagogastrostomy as advocated by Heyrovsky[30] is a side-to-side anastomosis between the dilated portion of the esophagus and the fundus of the stomach; (2) cardioplasty as advocated by Wendel[31] consists of a longitudinal incision in the lower part of the esophagus and the cardiac end of the stomach through the region of obstruction. This incision is closed in a transverse direction to enlarge the opening between the lower end of the esophagus and stomach; (3) a combination esophagogastrostomy and cardioplasty was advocated by Gröndahl[32] in which an anastomosis is made between the lower part of the esophagus and cardiac end of the stomach after a U-shaped incision is made through the constricted region of the esophagus and cardia; (4) extramucous esophagocardiomyotomy as advocated by Heller[33] consists of an incision down to the mucous membrane in the longitudinal circulatory muscles in the region of the obstruction; (5) cardiectomy and resection of the lower part of the esophagus and esophagogastric anastomosis may be used.[34, 35] For the first four procedures the abdominal approach usually is used, and, for the last procedure, the transthoracic approach.

SURGICAL EXPERIENCE AT THE MAYO CLINIC

Twenty-three patients have been treated surgically at the clinic for cardiospasm, and most of the operative procedures just mentioned have been utilized.

One patient had two operations, making twenty-four operative procedures by several different surgeons (Fig. 6). Many of these twenty-three cases have been reported in the literature. Plummer[1] in 1906 reported a retrograde transgastric dilatation performed by W. J. Mayo in 1905; Judd, Vinson, and Greenlee[36] reported two cases in 1929; Craig, Moersch, and Vinson[37] reported one case in 1934; Gray and Skinner[38] reported seven cases in 1940, including the previously reported cases in which operation had been performed at the clinic up to that time. Clagett, Moersch, and Fischer[39] reported four cases in 1945.

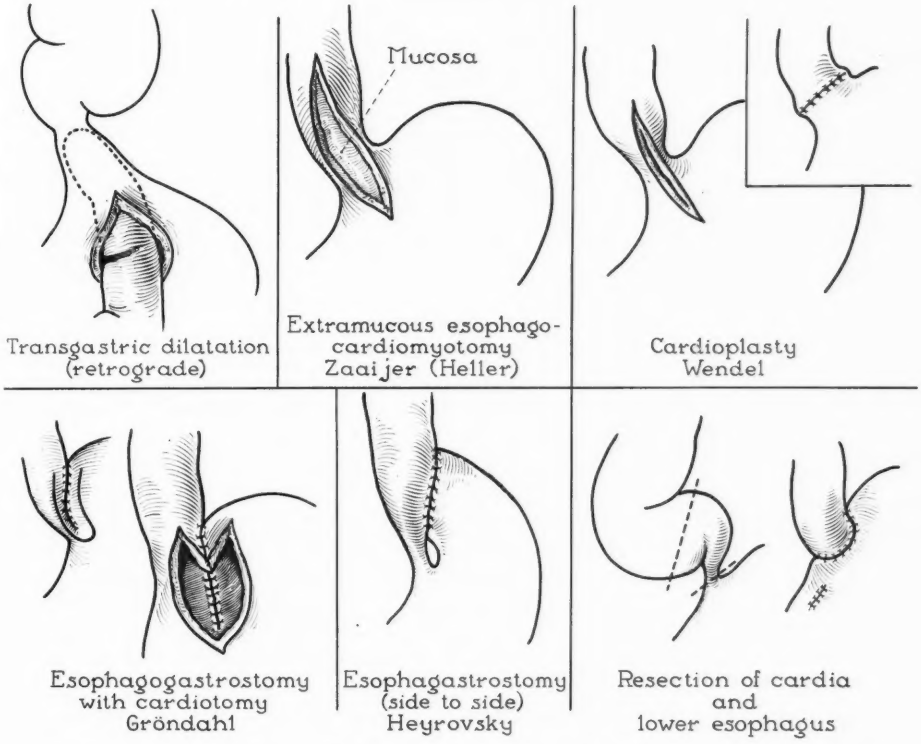

Fig. 6.—Various types of operative procedures performed in this series of twenty-three patients.

The chief indication for surgical intervention in most of our twenty-three cases was an unsatisfactory result from dilatation or other conservative methods of treatment. In many of our cases, dilatation and thickening of the walls as well as elongation of the esophagus were present. These marked secondary changes are usually associated with the more severe types of cardiospasm. We believe that the results of surgical treatment are more likely to be unsatisfactory when these marked secondary changes are present. This, however, is only one of the factors associated with unsatisfactory results from operative treatment. There is increasing evidence to indicate that the type of operative procedure

is important in the long-term results. Most procedures permit regurgitation of gastric secretions which cause inflammatory changes in the lower part of the esophagus.

The operative procedures utilized for our twenty-three patients are shown in Fig. 6. Four patients were treated by transgastric retrograde dilatation, one by sympathectomy* and one by both retrograde dilatation and sympathectomy. Of these six patients, five had poor results and one patient who had retrograde dilatation alone obtained a good result. The only operative death in the twenty-three cases followed transgastric retrograde dilatation. The patient died of pneumonia five weeks following the dilatation. These procedures have not been done in recent years.

In four of the twenty-three cases esophagogastrostomy of the Heyrovsky type was used. Results were poor in two cases and good in two. All four of these patients had marked dilatation of the esophagus before operation, and three had retention of food. Symptoms had been present for two to twenty-five years before operation. One patient was 26 years of age, and three were more than 60 years of age. Of the two patients who obtained poor results, one, aged 69 years, had had symptoms for twenty-five years and had been treated by twelve dilatations. This patient died four years after operation from hemorrhage resulting from esophagitis and ulceration. The other was 26 years of age and had had symptoms for two years. Operation relieved the symptoms for one year, and then symptoms of obstruction and esophagitis recurred.

In five cases, esophagogastrostomy of the Gröndahl type was employed. Results were poor in three cases, fair in one, and good in one case. The ages of these patients were from 20 to 57 years, and symptoms had been present for nine to twenty-four years. All five had enlargement of the esophagus, and three had angulation with retention. One patient who had a poor result was 31 years of age, and had had symptoms for nine years (Case 1). Another was relieved for three years, and then symptoms recurred. Six and one-half years after the first operation a second operation, transthoracic cardiectomy, was performed. Since the second operation four months ago, symptoms have been relieved. The third patient who obtained a poor result from this operation had a markedly enlarged and elongated esophagus with marked retention of food (Case 2).

In four of the twenty-three operative cases, cardioplasty of the Wendel type was carried out. Results were good in one case, fair in two, and poor in one. Of the last three patients, one had symptoms of esophagitis on examination six years after operation, one was having attacks of dysphagia six years following operation, and the third obtained no relief from the operation.

In two of the twenty-three cases, transthoracic resection of the lower part of the esophagus and cardia with esophagogastric anastomosis was carried out. In one case the operation was done four days after rupture of the esophagus following an esophagoscopic examination. Five and one-half years have now elapsed since the operation, and results are good. In the other case the operation was carried out six years after esophagogastrostomy of the Gröndahl type which

*This case reported by Craig, Moersch, and Vinson[37] as a case of cardiospasm has been reclassified and is now considered a case of diffuse spasm of the esophagus.

did not relieve symptoms. As only four months have elapsed since the second operation it is too soon to evaluate the result, but thus far the patient has had a good result.

In the remaining three of the complete group of twenty-three cases, esophagocardiomyotomy of the Heller type was used. Results were considered good in one case (Case 3). In the other two the operation was performed within the last eight months, and it is too soon to determine anything but the immediate result. This, however, has been good in both cases. A transthoracic approach was used in two cases, and an abdominal approach in one case.

CASE REPORTS

Four cases in which one of us (Harrington) performed the operations are reported.

CASE 1.—A man, aged 31 years, was first admitted to the clinic Jan. 25, 1942. For nine years symptoms of dysphagia, with substernal pain, regurgitation, and vomiting, as well as retention of food in the esophagus had become progressively more severe. He lost 15 pounds (6.8 kg.). Dilatation of the esophagus elsewhere had given some temporary relief.

Roentgenographic examination of the esophagus showed cardiospasm with obstruction at the cardia, marked enlargement and tortuosity of the esophagus, and retention of large amounts of food.

Hydrostatic dilatation was done in February, 1942, without relief of symptoms.

On February 21 esophagogastrostomy with cardiotomy (Gröndahl type) was performed. The patient had no difficulty in swallowing food after operation, but had a burning in the lower part of the esophagus which was relieved by alkalies. He continued to have burning pain low in the esophagus and some, but not severe, attacks of dysphagia. Esophagoscopic examination six months after operation showed esophagogastritis with ulceration low in the esophagus. The patient has returned several times since and has had two courses of dilatations without any improvement of symptoms.

Nine years after operation the patient wrote that his general health was good, he was working every day, but had more or less constant burning in the esophagus after meals which was relieved by alkalies. He also was having periodic attacks of dysphagia and vomiting. He said, "I am grateful for the operation as I get along a thousand times better than before the operation."

We, however, consider the result in this case poor. The improvement in subjective symptoms was due to the release of the obstruction by the operation but this type of operative procedure employed has permitted the gastric secretions to flow into the esophagus. The ensuing esophagitis is the chief cause of his present symptoms. However, some patients have obtained relief of symptoms from this type of operative procedure. It is possible, therefore, that surgical treatment might have been more effective if it had been done before the secondary changes due to dilatation and retention of food had developed in the esophagus.

CASE 2.—A woman, aged 45 years, was first admitted to the clinic Oct. 5, 1939, because of attacks of dysphagia, regurgitation, and vomiting of food. The number and severity of these attacks had been increasing, and at this time she was vomiting large quantities of food every night.

The roentgenogram showed marked dilatation and obstruction at the cardia and cardiospasm (Fig. 7, *a*). A satisfactory esophagoscopic examination could not be done until two and one-half liters of food and secretions were removed from the markedly dilated and tortuous esophagus. After repeated unsuccessful attempts, a thread was passed (through a

stomach tube) through the obstruction at the cardia and the esophagus was dilated over the thread with a 60 F. sound. Repeated attempts to pass the esophagoscope over the thread through the cardia into the stomach were unsuccessful. Because it was impossible to treat the obstruction at the cardia by dilatation methods, surgical treatment was advised and refused at that time.

The patient returned Sept. 3, 1948, and stated that unsuccessful attempts had been made in her home locality to dilate the esophagus both from above and below. In 1940 gastrostomy had been done for feeding and when the opening was closed symptoms recurred. They had become progressively more severe and she had lost 50 pounds (22.7 kg.). Roentgenographic and esophagoscopic examinations showed a markedly enlarged tortuous esophagus with obstruction at the cardia. Four liters of foul material were aspirated from the esophagus. Oct. 18, 1948, esophagogastrostomy (Gröndahl type) with cardiotomy was done through an abdominal incision (Fig. 6).

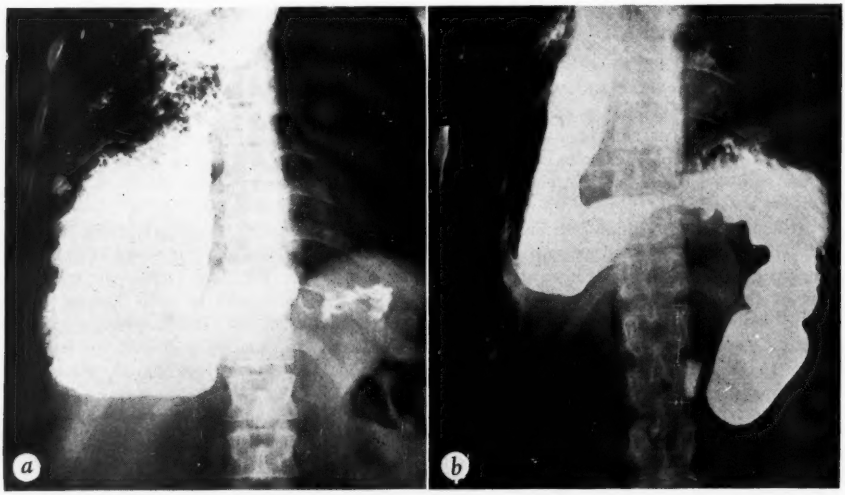

Fig. 7 (Case 2).—*a*, On admission cardiospasm with extreme dilatation and elongation of esophagus. Marked retention of food and secretions. Esophagus occupies most of right half of the thorax. *b*, Three months after abdominal esophagogastrostomy with cardiotomy (Gröndahl). Dilatation of esophagus is less marked.

After the operation the dysphagia and retention of material in the esophagus lessened (Fig. 7, *b*), but daily aspiration of the esophagus was continued. Improvement, however, was only temporary for retention of large quantities of food, pain below the sternum and to the right shoulder developed and severe hemorrhages occurred. Resection of the lower part of the esophagus and cardia of the stomach and vagus nerves was performed elsewhere in November, 1949.[27] A few weeks after this operation, she had massive hematemesis. In December, 1949, the antrum of the stomach was resected elsewhere. Convalescence was complicated and she died five months later.[27]

This case shows the serious complications that can arise from cardiospasm as well as the difficulty that may follow treatment after these complications have developed. We believe that when elongation and retention of secretions are present the danger of esophagitis complicating any type of operation which permits gastric juice to enter the esophagus is greater than in cases in which these changes have not occurred. The surgical problem in these cases is not only the removal of the obstruction, but also correction of the elongation of the

esophagus which acts as a reservoir for food and secretions. These patients may be best treated surgically by resection of the elongated portion of the esophagus with esophagogastric anastomosis. However, it would be better to institute surgical treatment early in the course of the disease and thus avoid the necessity for these more extensive operative procedures.

CASE 3.—A woman, aged 30 years, came to the clinic Jan. 26, 1949. She had had intermittent and progressively increasing difficulty in swallowing since childhood. During the past year she had had attacks of severe dysphagia, vomiting, and spasm of the lower part of the esophagus as well as pain between her shoulder blades.

Esophagoscopic and roentgenographic examinations at the clinic showed marked dilatation of the upper third of the esophagus with a fluid level due to pouching of the wall of the esophagus but no opening suggestive of a diverticulum, and moderate dilatation of the lower two-thirds of the esophagus with narrowing of the cardia and some associated diffuse spasm. Hydrostatic dilatations were carried out from Feb. 9 to 17, 1949, without relief of symptoms.

The patient returned to the clinic in April, 1949, because her symptoms were more marked than before dilatation. Surgical treatment was advised. She was told that we could not promise complete relief of all of her symptoms inasmuch as they were due to diffuse spasm of the entire lower part of the esophagus as well as to cardiospasm. We thought relief of the obstruction at the cardia might lessen her symptoms.

On May 4, 1949, an extramucous esophagocardiomyotomy (Zaaijer modification of the Heller operation) was carried out with a left transthoracic approach.

The immediate result was entirely satisfactory in that dysphagia was relieved. Roentgenographic studies at the time of her dismissal revealed some spasm of the lower part of the esophagus. When she returned for further examination one year later, she had been completely relieved of the obstructive symptoms and was able to eat all types of food without difficulty. However she occasionally had attacks of mild spasm associated with some pain between her shoulder blades when emotionally upset or under stress. Two years after the operation (February, 1951) she wrote that her condition was essentially the same except for some gradual improvement.

Symptoms in this case were due to cardiospasm and diffuse spasm of the lower part of the esophagus. Extramucous esophagocardiomyotomy relieved the obstructive symptoms due to the cardiospasm but, as expected, some of the symptoms due to the spasm of the lower part of the esophagus have continued. This case exemplifies the importance of determining clinically whether the condition is cardiospasm or if there is also an associated spasm of the lower part of the esophagus as the operative treatment for cardiospasm will not correct the diffuse spasm of the lower part of the esophagus.

CASE 4.—A man, aged 23 years, first came to the clinic Nov. 9, 1945, because of regurgitation of food and coughing and choking at night. For approximately two years he had had episodes of obstruction on swallowing food associated with pain at the lower end of the sternum. These attacks had become more frequent and severe.

Roentgenograms of the esophagus and stomach revealed cardiospasm with moderate dilatation of the esophagus. One dilatation completely relieved him for two months. Then his previous symptoms gradually reappeared and became more severe.

He returned to the clinic in April, 1947. The roentgenographic appearance of the esophagus was essentially the same as at the previous visit. Hydrostatic dilatations of the esophagus on April 14, 1947, relieved his symptoms for nearly a year.

He returned again Feb. 22, 1951. The roentgenogram taken at this time showed slight dilatation of the esophagus (Fig. 8, *a*). Esophagoscopic examination showed a moderate degree of esophagitis in the lower half of the esophagus but no ulceration. On completion of his examination, operative treatment was advised because he had received only temporary relief from the two previous dilatations, the second of which was a thorough hydrostatic

procedure, and it was thought that further dilatations would not give more than temporary relief. Accordingly, on March 2, 1951, a Zaaijer modification of the Heller extramucous esophagocardiomyotomy was done with a left transthoracic approach.

The immediate result was satisfactory. Symptoms were relieved and the roentgenogram (Fig. 8, *B*) before dismissal revealed a normal esophagus with no obstruction at the cardia.

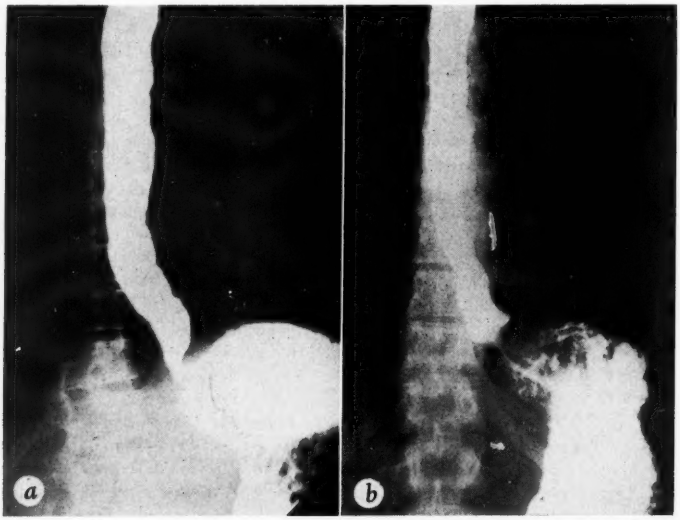

Fig. 8 (Case 4).—*a*, On third admission after two previous dilatations cardiospasm with some dilatation of esophagus; *b*, three weeks after extramucous esophagocardiomyotomy; no obstruction at cardia and esophagus is normal.

In this case surgical treatment was instituted early before the secondary changes and retention had occurred. We believe this is the best course to follow in treatment of cardiospasm which does not respond to dilatation. We are hopeful that the extramucous esophagocardiomyotomy will give complete relief. We hope, too, that it will prevent the esophagitis which so frequently follows a direct anastomosis between the esophagus and stomach when there is no sphincteric control to prevent the regurgitation of gastric secretions into the esophagus.

TECHNIQUE OF EXTRAMUCOUS ESOPHAGOCARDIOMYOTOMY EMPLOYED

The extramucous esophagocardiomyotomy of the Heller type employed in our cases is the modified procedure of Zaaijer,[40] which is advocated by Maingot[41] and others.[42-45] This technique and the transpleural approach used in the two last cases reported in this series will be described (Fig. 9).

A left posterolateral incision is made over the eighth rib on the left and the posterior two-thirds of the rib is resected. The pleural cavity is entered through the posterior periosteum of the eighth rib. The suspensory ligament of the lower lobe of the left lung is incised and the lung is retracted. The mediastinal pleura over the esophageal ring and the lower part of the esophagus is incised and the esophagus is elevated into the pleural cavity. The attach-

ments of the diaphragmatico-esophageal membrane at the esophageal ring are separated from the lower part of the esophagus and cardia by blunt dissection. The esophageal ring is incised and the cardiac end of the stomach is brought into the pleural cavity. The entire constricted area of the lower part of the esophagus and cardia of the stomach are exposed and straightened out by traction. The vagus nerve is retracted to the inner side. An incision is made upward through the anterior surface of the longitudinal and circular muscles of the constricted portion of the esophagus well into the dilated portion of the esophagus and downward through the circular muscle of the cardia and muscle

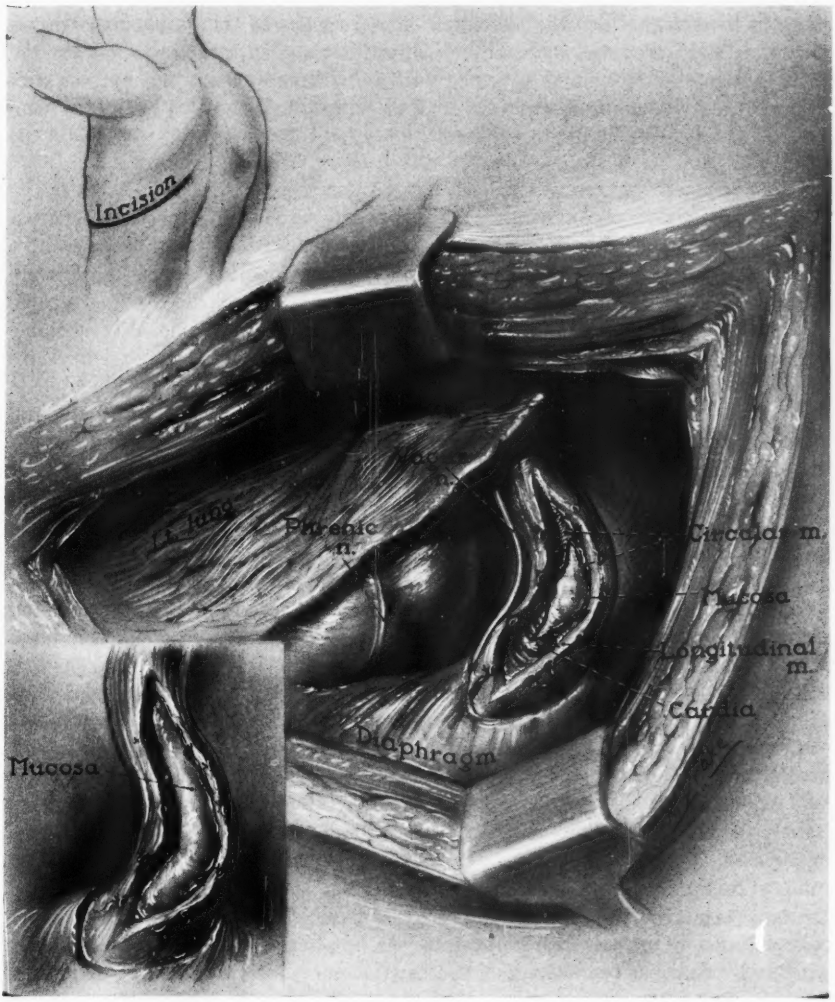

Fig. 9.—Technique of extramucous esophagocardiomyotomy (Zaaijer modification of the Heller procedure), instituted in Cases 3 and 4. Transthoracic approach.

walls of the stomach to the mucous membrane. The circular layers of muscle of the esophagus and cardia are usually very thin and often somewhat adherent to the underlying mucous membrane at the cardia. This circular layer must be separated with great care to avoid perforation of the mucosa. The blood vessels are larger and more numerous in the cardia and stomach than in the esophagus. Many of these vessels require a transfixing suture. The length of the incision depends on the amount of narrowing and constriction of cardia and lower part of the esophagus but is at least 10 cm. The mucous membrane is permitted to protrude throughout the entire extent of the longitudinal incision through the constricted area of esophagus and cardia. The cardiac end of the stomach is replaced into the abdomen. The incision in the esophageal ring is sutured with interrupted silk and the incision in the mediastinal pleura over the lower part of the esophagus is closed with interrupted catgut. The chest wall is closed and the pleural cavity is drained with an air-tight catheter and controlled suction for two to three days.

COMMENT ON SURGICAL RESULTS

From a review of our twenty-three surgical cases it is impossible to draw any definite conclusions concerning the best operative procedure. Twenty-three cases is a relatively small group and eight different operative procedures were utilized. The different procedures were used in from one to five cases each. These numbers are not only too small but the cases are not sufficiently uniform to compare the results of the different operations. However, certain deductions are justifiable: (1) The best results were obtained in those cases in which there were only moderate secondary changes of dilatation and thickening of the wall of the esophagus. (2) The poorest results were obtained when there was marked dilatation especially when it was associated with retention and elongation of the esophagus regardless of the type of operative procedure.

Comparison of the results revealed that transgastric retrograde dilatation or sympathectomy or both, almost always gave unsatisfactory results (poor in five cases and good in one). These procedures have not been utilized in recent years. The other five types of operative procedures utilized in the remaining eighteen cases represent the operations for cardiospasm most frequently used at this time. The results obtained from these five different operations in this series of eighteen patients were varied but essentially the same with each type of operation with the probable exception of the esophagocardiomyotomy. Some of the patients who have undergone esophagocardiomyotomy have not been operated on a sufficient length of time to permit a final evaluation of the results.

All of these operative procedures are directed toward relieving the obstruction. The immediate and early result in practically all cases regardless of type of operative procedure was satisfactory and the patient was greatly relieved because of the removal of the obstruction. In 6 of these cases, however, the long term result was unsatisfactory because of esophagitis from the free regurgitation of gastric secretions into the esophagus. In the three cases in which the result has been classified as fair, the esophagitis was of a lesser degree. The effect of gastric secretions in the esophagus has been discussed repeatedly. Maingot,[41] Barrett and Franklin,[43] and others[27, 46] have called attention to the

fact that the late unfavorable results after operations for cardiospasm are due to the development of peptic ulceration in the esophagus. Late fatal hemorrhages occurred in two patients, four years and one and one-half years after operation.

The development of dysphagia in the other patients who had recurrent trouble is presumably the result of inflammatory and cicatricial changes produced by the regurgitation of gastric secretions. The mechanism is not unlike that described by Allison,[47] Olsen and Harrington[48] and others, in which incompetence of the cardiac sphincter leads to peptic esophagitis, short esophagus and esophageal stricture.

The ideal surgical operation is one in which the cardiac sphincteric control of gastric secretions will be maintained and the obstruction of the esophagus relieved. Our experience at this time with the esophagocardiomyotomy (Heller type of procedure) is not sufficient in number of cases or length of time since the operations to permit any final conclusion. The results so far, however, have been encouraging. We are hopeful that this procedure will accomplish the objective of operative treatment for cardiospasm which is the relief of the obstruction without superimposing a complication such as esophagitis that may be as serious as or more dangerous than the original condition.

COMMENT AND CONCLUSIONS

The cause of cardiospasm is not known and hence a truly rational approach to the treatment of this disease has not been evolved. Whether there is actual spasm at the physiologic sphincter of the lower part of the esophagus or whether the cardia merely fails to open, is debatable. Whatever the etiology of this disease may be, the present methods of treatment are directed toward the relief of obstruction at the cardia.

Our study and experience with 601 cases have led us to formulate certain opinions and conclusions. These are as follows:

1. Exact diagnosis is important. Cardiospasm must be differentiated from diffuse spasm of the esophagus. Diffuse spasm does not respond favorably to either hydrostatic dilatation or surgical treatment.

2. Hydrostatic dilatation at present is the treatment of choice for most patients with cardiospasm. Relief of dysphagia for periods of four to sixteen years was obtained from a single course of dilatation in 60.1 per cent of our 452 traced cases. If we are given the opportunity of treating those patients a second time for whom an initial course of treatment by hydrostatic dilatation did not permanently relieve dysphagia, the percentage of satisfactory results can be increased to 75 per cent. However, if satisfactory results are not obtained with two courses of hydrostatic dilatation, additional treatment is likely to be of little avail. We must anticipate that about 20 per cent of the patients cannot be permanently relieved by dilatation.

3. Successful dilatation in the case of properly diagnosed cardiospasm depends on (a) accurate placement of the dilator in the cardia, and (b) severe forceful dilatation of the physiologic sphincter by an expanding dilator. A previously swallowed thread permits accurate placement of the dilator and adds to the safety of the procedure.

4. Surgical treatment should be considered for those patients who fail to respond to two or three courses of dilatation therapy. Every effort should be made to carry out surgical treatment before marked changes in the esophageal wall have taken place. Less favorable results can be expected when surgical therapy of the advanced cases is undertaken.

5. Surgical correction of cardiospasm may destroy the sphincteric property of the cardia. The regurgitation of gastric secretions may result in peptic esophagitis and account for poor surgical results.

6. The Heller operation appears to be the most advantageous surgical procedure. This procedure permits the cardia to retain some of its sphincteric property.

7. If the Heller operation is not feasible and one of the operations is performed which destroys the sphincteric action of the cardia, then an effort must be made to reduce the acid-production of the stomach.

8. The results of the surgical management of cardiospasm at the clinic have not been very favorable for two reasons: Most of our patients selected for surgical treatment had far advanced cardiospasm for which dilatation therapy was impossible or had failed to give relief. Most of the operative procedures carried out permitted reflux of gastric secretions into the esophagus. The passage of time has given us an opportunity to determine the late results in most of our cases. Peptic esophagitis and ulceration were responsible for most of the poor results.

9. Treatment of cardiospasm, either by hydrostatic dilatation or by surgical means, can be carried out with little risk.

REFERENCES

1. Plummer, H. S.: Cardiospasm, With Report of Cases, J. Minnesota State M. A. and Northwestern Lancet **26**: 419-424, 1906.
2. Plummer, H. S.: Cardiospasm; With a Report of Forty Cases, J. A. M. A. **51**: 549-554, 1908.
3. Plummer, H. S.: Diffuse Dilatation of the Esophagus Without Anatomic Stenosis (Cardiospasm); a Report of Ninety-one Cases, J. A. M. A. **58**: 2013-2014, 1912.
4. Plummer, H. S., and Vinson, P. P.: Cardiospasm; a Report of 301 Cases, M. Clin. North America **5**: 355-369, 1921.
5. Vinson, P. P.: The Diagnosis and Treatment of Cardiospasm, J. A. M. A. **82**: 859-861, 1924.
6. Moersch, H. J.: Cardiospasm in Infancy and in Childhood, Am. J. Dis. Child. **38**: 294-298, 1929.
7. Moersch, H. J.: Cardiospasm: Its Diagnosis and Treatment, Ann. Surg. **98**: 232-238, 1933.
8. Moersch, H. J.: Die Behandlung des Kardiospasmus, Arch. klin. Chir. **186**: 456-460, 1936.
9. Moersch, H. J.: The Treatment of Cardiospasm, Gastroenterology **12**: 520-521, 1948.
10. Schmidt, H. W.: Cardiospasm in a Six Year Old Child, Proc. Staff Meet., Mayo Clin. **11**: 767-768, 1936.
11. Vines, R. W., and Olsen, A. M.: Cardiospasm With Associated Pellagra: Report of Case, Proc. Staff Meet., Mayo Clin. **18**: 389-395, 1943.
12. Moersch, H. J., and Camp, J. D.: Diffuse Spasm of the Lower Part of the Esophagus, Ann. Otol., Rhin. & Laryng. **43**: 1165-1173, 1934.
13. Schmidt, H. W.: Diffuse Spasm of the Lower Half of the Esophagus, Am. J. Digest. Dis. **6**: 693-700, 1939.
14. Penner, Abraham, and Druckerman, L. J.: Segmental Spasms of the Esophagus and Their Relation to Parkinsonism, Am. J. Digest. Dis. **9**: 282-287, 1942.
15. Russel, J. C.: Diagnosis and Treatment of Spasmodic Stricture of the Oesophagus, Brit. M. J. **1**: 1450-1451, 1898.
16. Tucker, Gabriel: Cardiospasm: A Pneumatic-mercury Dilator, Ann. Otol., Rhin. & Laryng. **48**: 808-816, 1939.

17. Browne, D. C., and McHardy, Gordon: Medical Management of Cardiospasm, New Orleans M. & S. J. **93:** 627-633, 1941.
18. Boros, Edwin: An Improved Esophageal Dilator, Arch. Otolaryng. **35:** 799-802, 1942.
19. Johnstone, A. S., and Wooler, G. H.: Cardiospasm, Overseas Post Grad. M. J. **3:** 399-410, 1949.
20. Vinson, P. P.: The Diagnosis and Treatment of Diseases of the Esophagus, Springfield, Illinois, 1940, Charles C Thomas, 224 pp.
21. Vinson, P. P.: Diagnosis and Treatment of Cardiospasm, South. M. J. **40:** 387-391, 1947.
22. Carr, D. T., and Vinson, P. P.: Cardiospasm, J. M. A. Georgia **27:** 440-445, 1938.
23. Seybold, W. D., Johnson, M. A., III, and Leary, W. V.: Perforation of the Esophagus; an Analysis of 50 Cases and an Account of Experimental Studies, S. Clin. North America **30:** 1155-1183, 1950.
24. Nagel, G.: Cardiospasm, California Med. **71:** 285-286, 1949.
25. Freeman, E. B.: Symptoms, Diagnosis and Treatment of Chronic Cardiospasm, Rev. Gastroenterol. **7:** 385-390, 1940.
26. Sifers, E. C., and Crile, George, Jr.: Cardiospasm; a Review of 100 Cases, Gastroenterology **16:** 466-474, 1950.
27. Fritz, J. M., Clark, D. E., and Adams, W. E.: The Diagnosis and Treatment of Cardiospasm, S. Clin. North America **31:** 173-187, 1951.
28. Ochsner, Alton, DeBakey, Michael, and DeCamp, P. T.: Surgery of the Esophagus, Ann. Otol., Rhin. & Laryng. **58:** 1171-1198, 1949.
29. von Mikulicz, J.: Zur Pathologie und Therapie des Cardiospasmus, Deutsche med. Wchnschr. **1** (pt. 1): 17-19, 1904.
30. Heyrovsky, Hans: Casuistik und Therapie der idiopathischen Dilatation der Speiseröhre. Oesophagogastroanastomose, Arch. f. klin. Chir. **100:** 703-715, 1913.
31. Wendel, W.: Zur Chirurgie des Oesophagus, Arch. f. klin. Chir. **93:** 311-329, 1910.
32. Gröndahl, N. B.: Cardioplastik ved Cardiospasmus, Nord. med. Ark. **49:** 236, 1916.
33. Heller, E.: Extramuköse Cardiaplastik beim chronischen Cardiospasmus mit Dilatation des Oesophagus, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **27:** 141-149, 1913.
34. Hoover, W. B.: Cardiospasm or Achalasia of the Cardia; Some Personal Observations and Practical Considerations With a Presentation of 7 Cases of Cardiectomy, Ann. Otol., Rhin. & Laryng. **59:** 766-802, 1950.
35. Womack, N. A., Brintnall, E. S., and Ehrenhaft, J. L.: Benign Obstruction of the Lower Esophagus, J. A. M. A. **145:** 283-288, 1951.
36. Judd, E. S., Vinson, P. P., and Greenlee, D. P.: Retrograde Dilatation of the Oesophagus for Cardiospasm, Surg., Gynec. & Obst. **48:** 494-497, 1929.
37. Craig, W. McK., Moersch, H. J., and Vinson, P. P.: Treatment of Intractable Cardiospasm by Bilateral Cervicothoracic Sympathetic Ganglionectomy: Report of Case, Proc. Staff Meet., Mayo Clin. **9:** 749-753, 1934.
38. Gray, H. K., and Skinner, I. C.: The Operative Treatment of Cardiospasm, J. THORACIC SURG. **10:** 220-236, 1940.
39. Clagett, O. T., Moersch, H. J., and Fischer, Albert: Esophagogastrostomy in the Treatment of Cardiospasm, Surg., Gynec. & Obst. **81:** 440-445, 1945.
40. Zaaijer, J. H.: Cardiospasm in the Aged, Ann. Surg. **77:** 615-617, 1923.
41. Maingot, Rodney: Surgical Treatment of Cardiospasm, Postgrad. Med. **5:** 351-360, 1949.
42. Barlow, D.: Treatment of Cardiospasm by Heller Type of Operation, With Special Reference to Choice of Operation, Its Indications and Technique, and With Report Upon 3 Personal Cases, Brit. J. Surg. **29:** 415-421, 1942.
43. Barrett, N. R., and Franklin, R. H.: Concerning the Unfavourable Late Results of Certain Operations Performed in the Treatment of Cardiospasm, Brit. J. Surg. **37:** 194-202, 1949.
44. Buckles, M. G.: Surgical Treatment of Cardiospasm, Am. J. Surg. n.s. **80:** 846-853, 1950.
45. Garin, H.: Modified Heller's Operation for Cardiospasm (Achalazia of Cardia), South African M. J. **18:** 382-383, 1944.
46. Schalm, L.: Late and Fatal Complication After Oesophagogastrostomy; (Operation of Heyrovsky), Arch. chir. neerl. **2:** 166-169, 1950.
47. Allison, P. R.: Quoted by Olsen, A. M., and Harrington, S. W.[48]
48. Olsen, A. M., and Harrington, S. W.: Esophageal Hiatal Hernias of the Short Esophagus Type: Etiologic and Therapeutic Considerations, J. THORACIC SURG. **17:** 189-207, 1948.

VARIATIONS IN THE BRONCHOVASCULAR PATTERNS OF THE RIGHT LOWER LOBE OF FIFTY LUNGS

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IN A previous article by Smith and Boyden (1949) the principal variations in the distribution and arrangement of the segmental bronchi were established, first by injections and then by dissection of injected specimens. At that time, even if the technique had permitted, it would not have been feasible to analyze the vascular patterns owing to their complexity and the necessity of first mastering the bronchial variations. Nevertheless, it was recognized that a knowledge of the arrangement of arteries and veins is as essential to segmental resection or lobectomy as familiarity with bronchial patterns. Indeed, the present study has shown that such malarrangements as the splitting of the medial basal bronchus by the basal branches of the inferior pulmonary vein may prevent the resection of that segment even though the bronchogram might not suggest the danger of resection to the uninitiated observer.

To obtain the necessary information, fifty right lower lobes were removed from cadavers and dissected completely. Accurate drawings were made of the paravertebral view of the medial basal segment *(B^7)*, of the dissected anterior surface of the lobe, of the lateral view—for optimum exposure of the arteries—and of the opened bronchial tree.

Obviously, it was necessary again to identify the bronchial variations. New information has been gained because dissection of soft cadaver material is more accurate than dissection of specimens filled with gelatin, and in addition percentages could be calculated on the basis of 100 specimens, those of Smith and Boyden's being added to our own.

THE GENERAL VENOUS PATTERN

Since the veins lie between adjacent bronchi and offer cleavage planes by which segments and subsegments can be separated from one another, it is advantageous to start with the venous pattern. With minor exceptions the veins of the right lower lobe drain into the inferior pulmonary vein. As shown in Plate 1—a diagram of the dissected anterior surface of a truncated right lower lobe—the inferior pulmonary has two principal tributaries, a *superior vein (V^6)* draining the superior segment, and a *common basal* vein draining the basal segments.* The latter is formed by the union of a *superior basal* vein, draining primarily segments B^8 and B^9, and an *inferior basal vein* draining

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Aided by grants from the Medical Research Funds of the Graduate School as part of a long-term program under the direction of E. A. Boyden.

Received for publication April 30, 1951.

*These correspond to the truncus dorsalis and truncus ventralis of Adachi (1933), and to the ramus superior and ramus inferior of Herrnheiser and Kubat (1936). Our terms are designed to emphasize the relation of these veins to the superior and the basal segments.

primarily segment B^{10} (or segments B^{9+10}). This pattern occurs in 78 per cent of specimens. The anomalous patterns found in the remaining 22 per cent will be discussed at the end of this section (Table III).

The varying composition of the basal veins is shown in Table I.

TABLE I. PATTERN OF BASAL VEINS

	SUP. BASAL	INF. BASAL	PERCENTAGE	
1.	Uncomplicated types			38%
	V^{8+9}	V^{10}	32%	
	V^{8}	V^{9+10}	6%	
2.	"Split" types			62%
	V^{8+9}	V^{9+10}	44%	
	$V^{8, 9, 10}$	V^{10}	14%	
	V^{8}	$V^{8, 9, 10}$	4%	

In the uncomplicated types all of V^8, or V^9 or V^{10} is found to be a tributary of either the superior or the inferior basal vein. The most common pattern (32 per cent) is that shown in Plate 1. In the "split" type, part of either V^8, or V^9 or V^{10} joins both the superior and inferior basal veins. Here, the commonest arrangement (44 per cent) and, therefore, the prevailing pattern for the lobe, is that in which there are two V^9s, one joining the superior basal, the other the inferior basal vein. Unfortunately, none of the split types are shown in the illustrations, but the tabular presentation indicates the futility of giving descriptive names to the larger tributaries (cf. Melnikoff, 1922, and Herrnheiser and Kubat, 1936).

At this point, it is desirable to point out that the main trunks of the basal veins lie deep to the anterior surface of the lobe and that the key to their dissection are the intersegmental branches of B^8b (Fig. A, Plate 2). These superficial veins separate the medial basal (B^7) from the anterior basal segment (B^8). If one dissects obliquely medialward along the plane of these veins to V^9 and V^{10}, the medial basal segment can be lifted medialward thus exposing the deeper veins and bronchi. If one were to apply to the median basal the principles of segmental resection recently developed by Overholt, Woods, and Ramsay, the superficially placed A^7 (medial basal artery) would be ligated first (Fig. A, Plate 2), then B^7 (the medial basal bronchus). As the cut ends are pulled forward, the small segmental veins of V^7 would be ligated and then the plane of separation from the deeper segments developed along the anterior surfaces of V^8b, V^9 and V^{10} (Fig. E, Plate 2).

The veins of the medial basal segment (V^7) have not been included in the above classification for the reason that they are small veins ranging from one to six in number. If there is only one, it usually empties into the inferior basal vein or its junction with the superior basal (Plate 1) or directly into the inferior pulmonary vein (Plate 2). When V^7 is multiple, the orifices of V^7a may be scattered along the superior basal vein, and those of V^7b along the inferior basal, or located upon either of the large veins (as in Plate 6, #41, A).

Similarly the veins from the subsuperior zones (V^* and VX^*) have not been included in the above table since they are variable and may empty into either

the proximal or more distal portions of the great veins. Using V^* as an example, it terminates in the twenty-eight specimens in which it occurred as follows: once on the inferior pulmonary, coordinate with V^6 (cf. Plates 2 and 3); twice on V^6c or V^6b; six times on the common basal; three times on the proximal portion of the superior basal; five times on the proximal portion of the inferior basal; seven times on the distal portion of the superior basal or its branches; and four times on the distal portion of the inferior basal or its branches. In sum, it empties into the proximal portions seventeen times, and into the distal portions eleven times. (Incidentally, the termination of V^* in the common basal was noted seven times by Adachi. He named this vein the distal truncus dorsalis in contrast to V^6 which he called the proximal truncus dorsalis.)

As to the course of these veins from the posterior sector of the lobe to the hilar region, one may say that almost always those which terminate in the proximal portions of the great veins pass medial to the posterior basal bronchus, B^{10} (cf. V,* lateral views, Plate 3, and #24, Plate 5). Those which terminate in the distal portions pass between B^8 and B^9 or between B^9 and B^{10} (cf. VX,* Plate 6, #41).

THE RELATION OF THE BASAL VEINS TO THE MEDIAL BASAL SEGMENT

Probably the basal segment which is least understood, clinically, is the medial basal (B^7). Doubtless this is due to the smaller size and the somewhat concealed position of its bronchus. In anteroposterior bronchograms it is hard to see. Brock (1945) has pointed out its arched appearance in lateral bronchograms, but few, if any, observers have paid attention to its branches, the anterior and medial rami (B^7a and B^7b). Yet, as a result of this scrutiny of fifty specimens, we have come to believe that it is the key to an understanding of the basal half of the lobe.

In analyzing the variations of this segment we have utilized a landmark which Dévé named "the hornlike projection of the cardiac segment." This is the anteroparavertebral ridge which separates the anterior from the paravertebral surfaces of the lobe. In preserved specimens, it occupies a position slightly medial to the medial boundary of the fossa of the inferior vena cava ("Ridge," Plate 4, Type II). Occasionally it coincides with that boundary. The relation of the pulmonary ligament to this ridge is variable. It may arise from the ridge or lie beyond it on the paravertebral surface. (In the latter case, one may speak of a "retro-pericardiac recess" between the ridge and the ligament.) Because the position of the ligament is variable it may not be used to define the anterior limit of the paravertebral surface. Posteriorly (Plate 7), that surface is limited by the costoparavertebral margin (Smith and Boyden).

Four types of the medial basal bronchus may be recognized (see diagrams, Plate 4): Type I, that in which B^7 is distributed to both anterior and paravertebral surfaces and passes superficial to the basal veins. This is the prevailing pattern (34 per cent); Type II, that in which the distribution is restricted to the anterior surface (22 per cent).* This represents only a slight variation

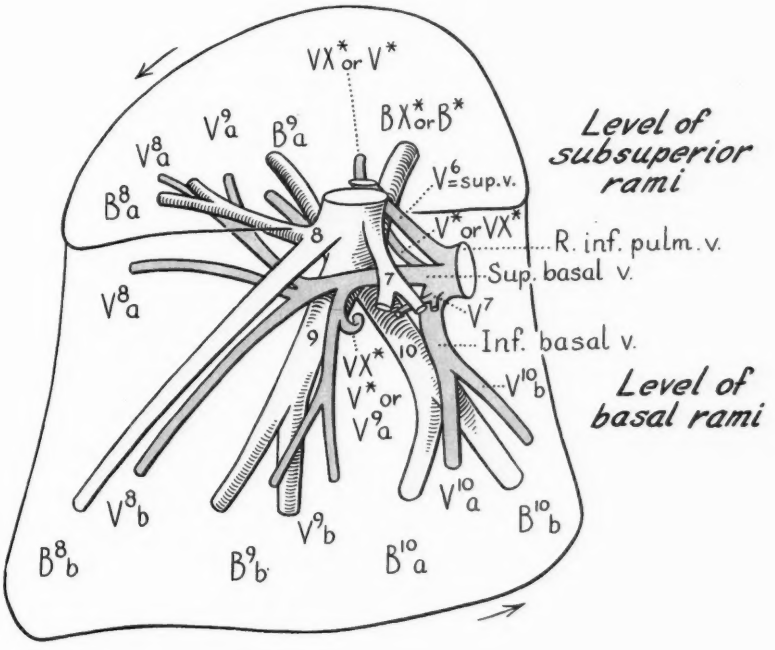
*In all four types deeper branches of the rami pass to the corresponding diaphragmatic surface, but for the purpose in hand, no further mention of these is necessary.

J. THORACIC SURG.
VOL. 22, NO. 2, AUGUST, 1951

EXPLANATION OF PLATES

(Sketches, by E. A. Boyden, rendered for publication by Lawrence B. Benson)

Plates 1-7.—A series of drawings illustrating variations found in the vascular patterns of the right lower lobe of fifty dissected lungs.



Scheme showing common arrangement of veins in basal segments

Plate 1.—Diagram of truncated right lower lobe to show venous pattern and method of numbering branches of basal veins. The vein on the counterclockwise side of a given bronchus (cf. direction of arrows) receives the number of that bronchus, viz., $V^{8}b$ is the vein on the counterclockwise side of $B^{8}b$. In the left lower lobe corresponding veins would lie on the clockwise side (Boyden, 1945). B^{7}, Medial basal bronchus; $B^{8}a$, $B^{8}b$, lateral and basal rami of anterior basal bronchus; $B^{9}a$, $B^{9}b$, lateral and basal rami of lateral basal bronchus; $B^{10}a$, $B^{10}b$, laterobasal and mediobasal rami of posterior basal bronchus; B^{*}, BX^{*}, subsuperior and accessory subsuperior bronchi.

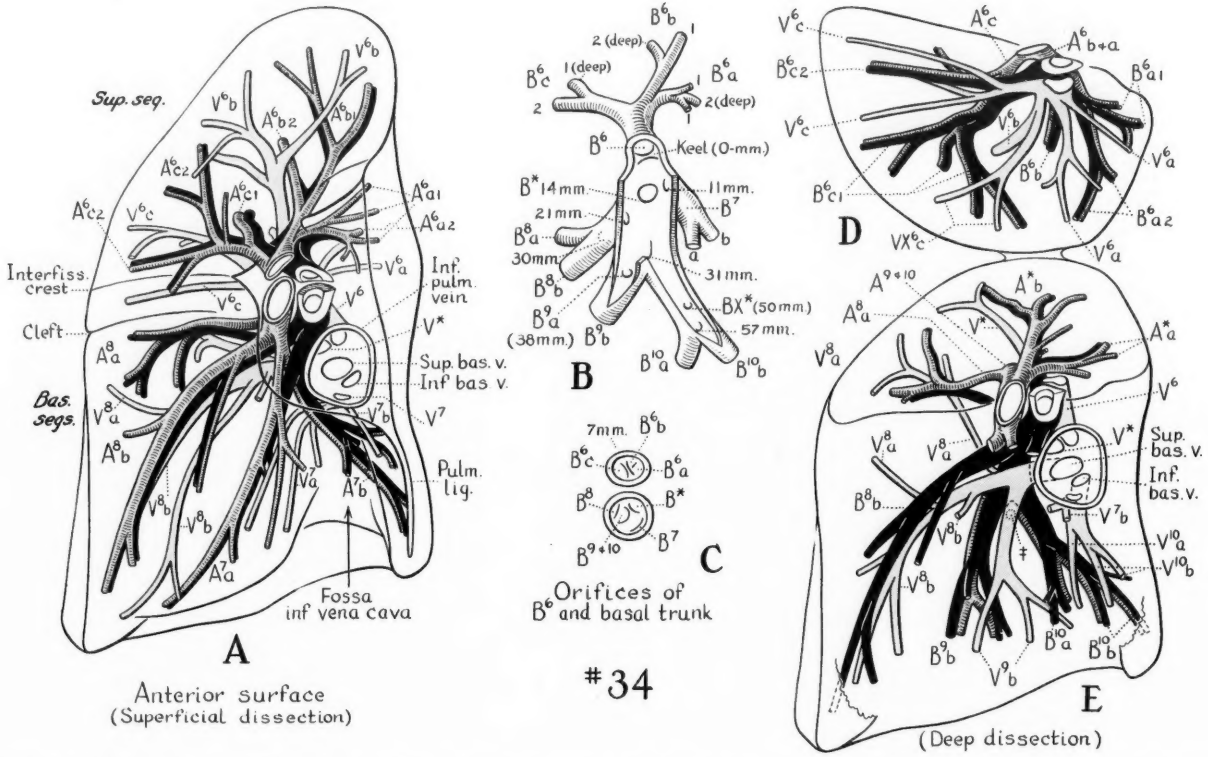


Plate 2.—Dissection of typical specimen (#34) classified as Type I (see Plate 4). Fig. *B*. opened bronchial tree; *C*, inverted bronchoscopic views; *D*, superior segment tilted back to show intersegmental veins. *Black* indicates bronchi; *red*, the pulmonary arteries; *blue*, the pulmonary veins. For names of segmental bronchi, see Text Figures 1 and 2 (cf. Boyden, 1949).

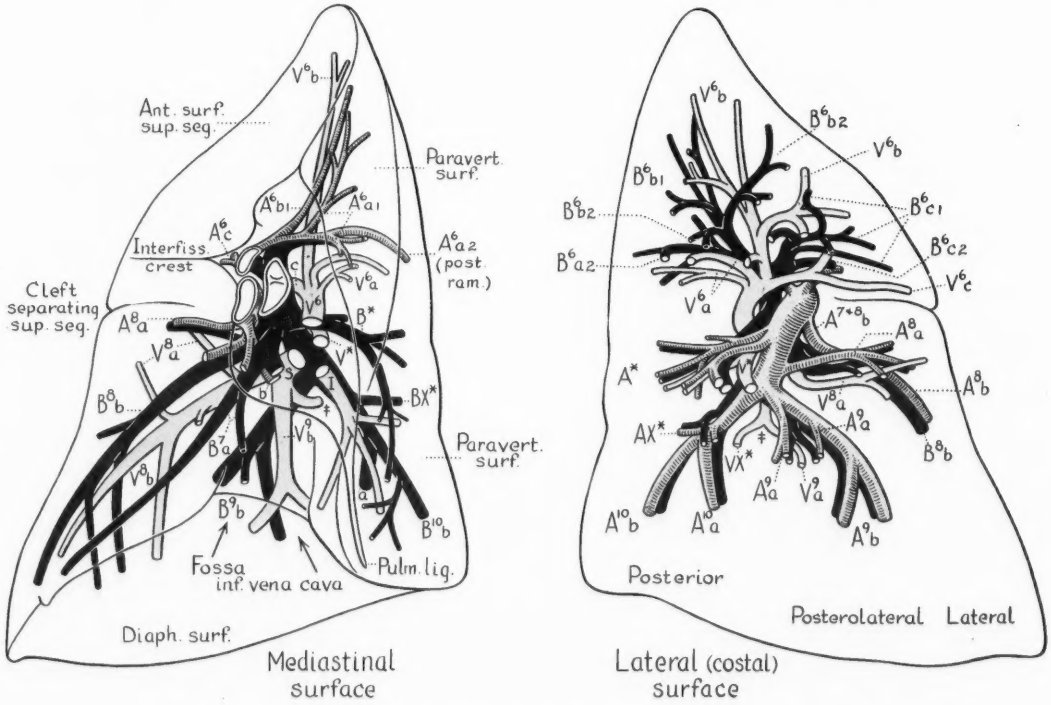


Plate 3.—Mediastinal and lateral views of the dissected specimen shown in Plate 2.

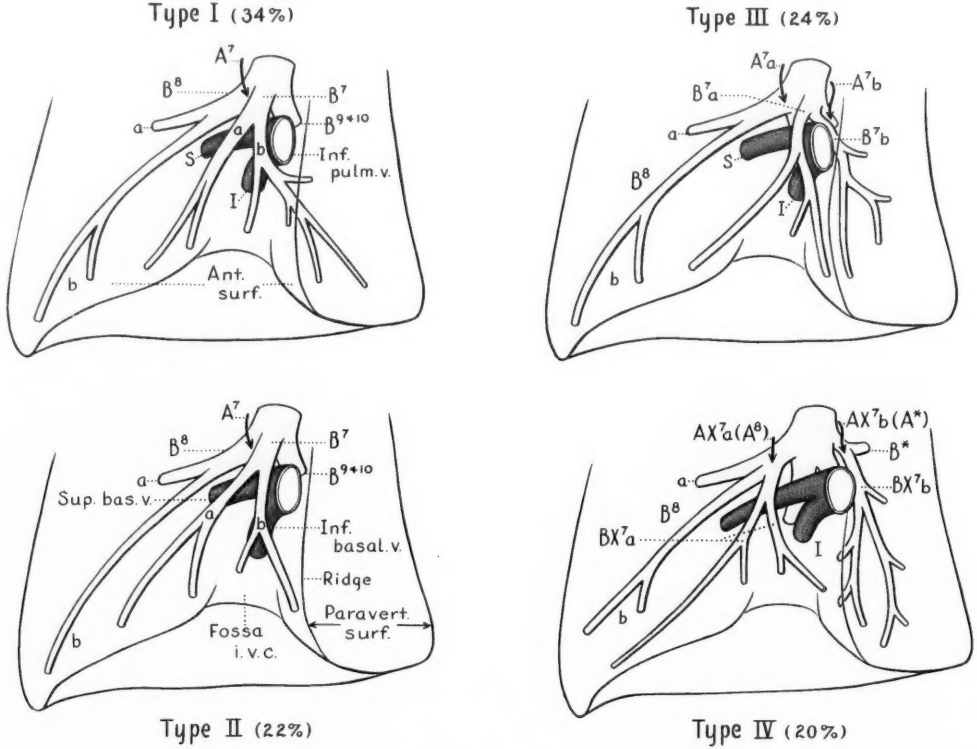
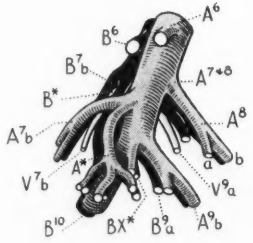
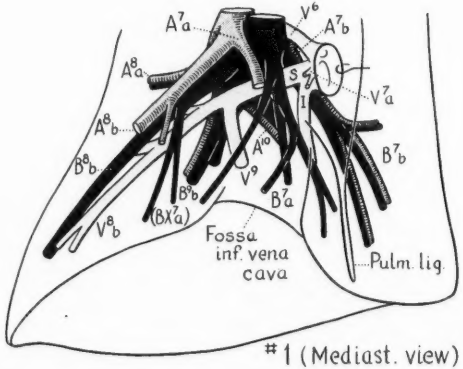


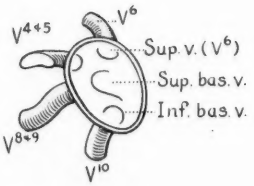
Plate 4.—Diagrams illustrating four types of arrangements of the branches of the medial basal bronchus, based on the origin and distribution of rami and their relation to the basal veins. Arrows indicate position of arteries. *B⁷a*, anterior ramus; *B⁷b*, medial ramus; *BX⁷a*, *BX⁷b* displaced anterior and medial rami.



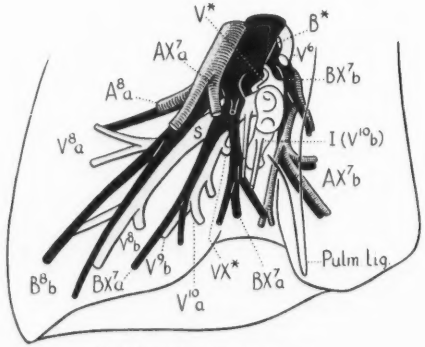
#1 (Lat. view)



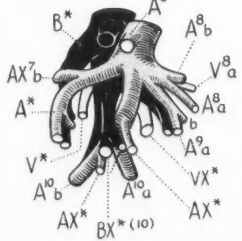
#1 (Mediast. view)



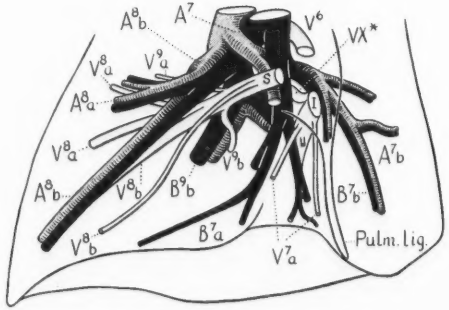
#10 (Inf. pulm. vein)



#24 (Mediast. view)



#24 (Lat. view)



#10 (Mediast. view)

Plate 5.—Sketches of three dissected specimens displaying arrangement of structures in specimens illustrating Type III (#1), a modified Type III (#10) and Type IV (#24). The vein drawn in upper right corner (#10) is not characteristic of Type III, but illustrates an unusual anomaly.

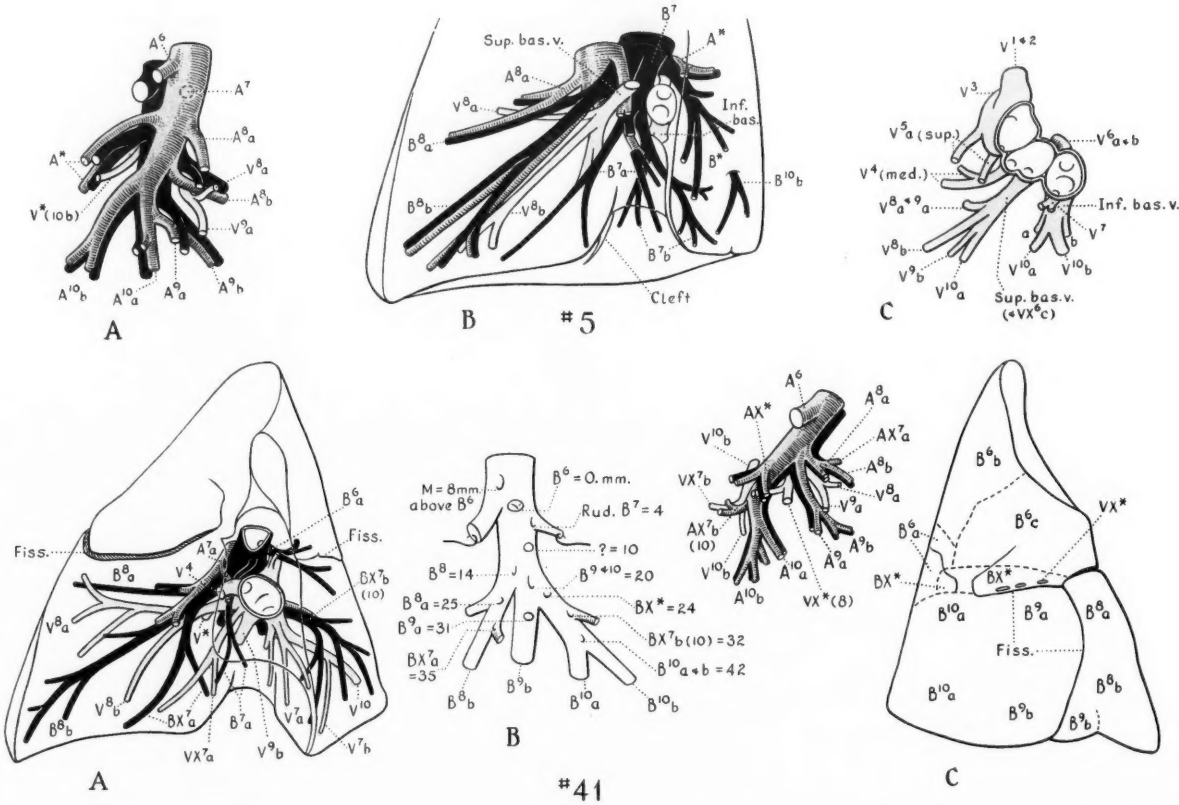


Plate 6.—Dissected specimens illustrating arrangement of structures in a modified Type I (#5) and in a type that is transitional between I and IV (#41). In both cases the inferior pulmonary vein is atypical. Fig. 41 C demonstrates that fissures do not necessarily separate segments but may develop between rami of a segment (e.g., $B^{9}b$). V^{*}, in Fig. A, should read VX^{*}.

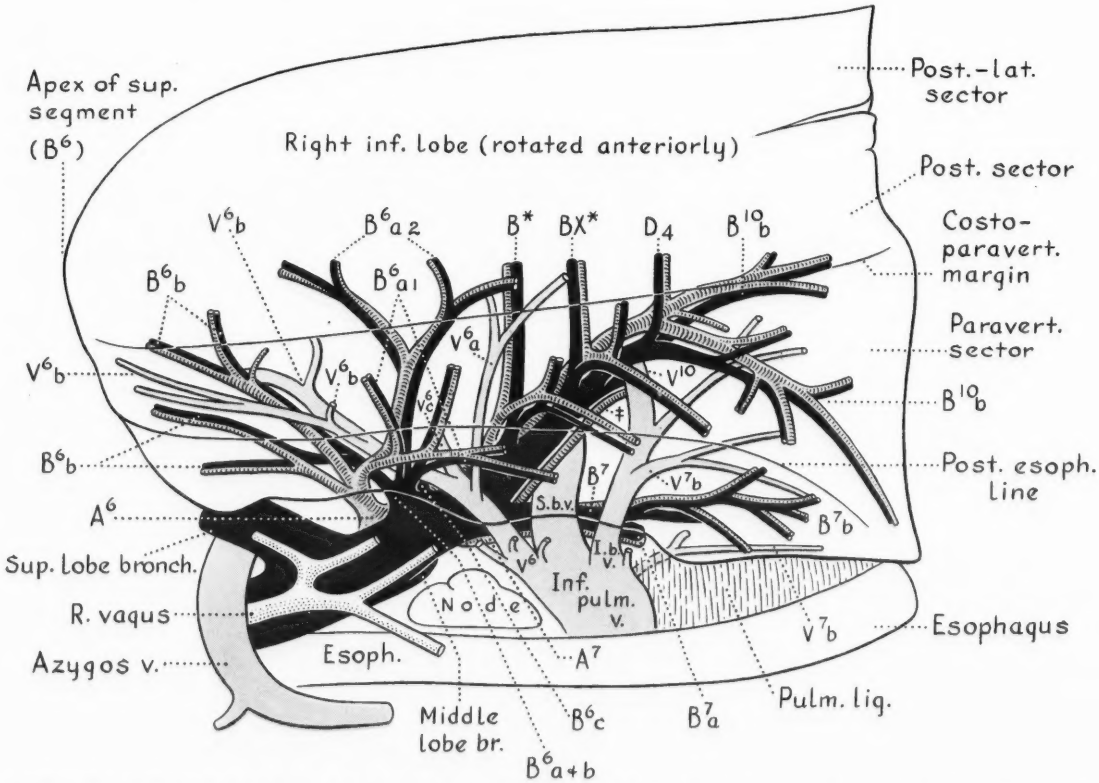


Plate 7.—Posterior surface of right lower lobe, dissected in situ after the lobe has been elevated (Specimen #14154). Note series of dorsal rami—B^6, B^*, BX^* and D_4 and their paravertebral branches. *S. b. v.*, superior basal vein; *I. b. v.*, inferior basal vein; ‡artery to $B^{10}a$ arising atypically from A^7.

from the normal pattern; Type III, that in which the bronchus arises from a more medial position, and bifurcates almost immediately around the basal veins (24 per cent). In this type the B^7a branch is superficial to the veins and is distributed anteriorly, the B^7b branch is deep to the veins and is distributed paravertebrally; Type IV, that in which B^7 is absent as such (20 per cent) and the branches which represent it arise independently from adjacent bronchi—BX^7a, from the anterior basal (B^8 or B^8b), and BX^7b from the subsuperior (B^* or BX^*) or, rarely from the posterior basal (B^{10}), as at #41 A, Plate 6.†

Exact drawings of specimens illustrating these types are shown in the following plates: Type I at A, Plate 2; a modified Type I at B, upper row of figures, Plate 6 (here the superior basal vein passes superficial to B^7, but the rest of the inferior pulmonary is normally situated, i.e., medial to the stem of B^7); Type III, upper row of Plate 5; a modified Type III, in lower right corner of Plate 5 (here, the superior basal vein passes superficial to B^7a, but the inferior basal passes between B^7a and B^7b); Type IV in the lower left corner of Plate 5. Finally, specimen *#41* in Plate 6 is of special interest as a transitional type. B^7 is represented by a rudimentary stem (B^7a). If this be ignored as negligible, then the specimen falls into Type IV with a BX^7a ramus arising from B^8b and a BX^7b ramus originating on B^{10}.

Before discussing the resectability of these types it is desirable to summarize the distribution of B^7. First, the line of division between B^7a and B^7b shifts in the different types (Plate 4). Prevailingly (Type I), it lies in the fossa of the inferior vena cava. In Type II, the line moves toward the lateral lip of the fossa, and in Types III and IV to the medial lip—specifically the anteroparavertebral ridge.

Second, the lateral limit of B^7, in Types I and II, was found to lie more than half way along the anterior margin from ridge to lateral margin of the lobe, in 42 per cent of specimens.

Third, B^7 was found to be absent as such in 20 per cent of specimens, to have a wholly anterior distribution in 22 per cent, and to supply both anterior and paravertebral surfaces in 58 per cent. Smith and Boyden's figures for fifty specimens were, respectively, 14 per cent, 8 per cent, and 78 per cent, but since in that article the anterior distance was measured from the variable pulmonary ligament, only the percentages for Type IV can be combined; that is, one can say that in 100 specimens B^7 was absent as such in 17 per cent.

From the foregoing study, it would appear that resection of the medial basal segment—either by itself, for localized bronchiectasis (Scannell), or in conjunction with middle lobectomy (Brock, 1950)—is technically feasible in Types I and II, but not in Type III. For in that type the basal veins lie between the rami of B^7 and ligation of them would seem to be fraught with hazard. Furthermore, in three-fourths of these specimens (as #1, Plate 5) the artery of B^7b originates posteriorly and can be reached only with difficulty.

The question thus arises as to how Type III may be identified preoperatively in bronchograms. Three criteria are submitted. First, B^7 originates more medi-

†The letter "X" is added to a ramus to indicate that it is displaced in origin and therefore accessory to the bronchus from which it does arise (Boyden, 1945).

ally than usual on the lower lobe bronchus. Second, it bifurcates almost at once into its principal rami.* Third, the fork is wider than usual in order to permit passage of the basal veins. Once these points have been established, segmental resection of the deeper ramus *(B^7b)* would seem to be inadvisable.

THE VEINS OF THE SUPERIOR SEGMENT *(V^6)*

The primary drainage of this segment is effected by the superior vein *(V^6)*. In 86 per cent of specimens it swings around the medial side of the lower lobe bronchus, between the superior and subsuperior bronchus *(B^7* and *B^**, Plates 3 and 7), to enter the inferior pulmonary vein on the medial side of *B^7* (Fig. *A*, Plate 2). In another 12 per cent it enters the inferior pulmonary at a lower level, either passing between *B^7a* and *B^7b* (6 per cent) or beneath *B^** (6 per cent), as in Plate 6 *(#5)*. Finally, in 4 per cent (2 per cent being included in the preceding category) the segment is drained by two separate veins *($V^6a + b$* and *V^6c; see items indicated by a dagger, in Table III).

In addition to this primary drainage there is frequently a secondary channel *(VX^6)* which drains into the posterior upper lobe vein *(V^3)*. This arises from the paravertebral or superior portion of the segment—an area that is frequently fused to the upper lobe. Boyden and Scannell found such a vein in 54 per cent of specimens.

The tributaries of *V^6* lie between the three rami of the superior segment and adjacent segments. Before discussing these it is necessary to review the bronchial patterns in the superior segment (see Fig. B, Plate 2). The variations are summarized in Table II, our figures having been combined with those of Smith and Boyden to give statistics based on 100 specimens.

TABLE II. SUMMARY OF THE SUPERIOR SEGMENTAL BRONCHUS *(B^6)*

	SMITH AND BOYDEN (50 SPEC.)	FERRY AND BOYDEN (50 SPEC.)	TOTAL (100 SPEC.)	
I. *B^6* arises as a single stem				
1. Bifurcates into	90%	88%		89%
$B^6a + b$ and *B^6c*	86%	72%	79%	
$B^6b + c$ and *B^6a*	--	8%	4%	
$B^6a + c$ and *B^6b*	--	2%	1%	
bizarre patterns	4%	6%	5%	
2. Trifurcates into *B^6a; B^6b; B^6c*	6%	4%		5%
II. *B^6* arises as two separate stems				6%
1. *$B^6a + b$; B^6c*	2%	6%	4%	
2. Miscellaneous patterns	2%	2%	2%	

A survey of Table II indicates that the prevailing pattern is that shown at B in Plate 2—namely, a bifurcation into *$B^6a + b$* and *B^6c*.

Three new observations have emerged from this study. The first records additional modes of bifurcation. The second establishes the almost constant presence of the deep branch of *B^6a* (98 per cent). Smith and Boyden reported it in only 38 per cent of specimens, but owing to the difficulty of dissecting specimens

*The average distance from the keel of *B^7* to the keel which separates the rami is 0.6 cm. In specimens of Types I and II, the average distance is 1.35 cm.

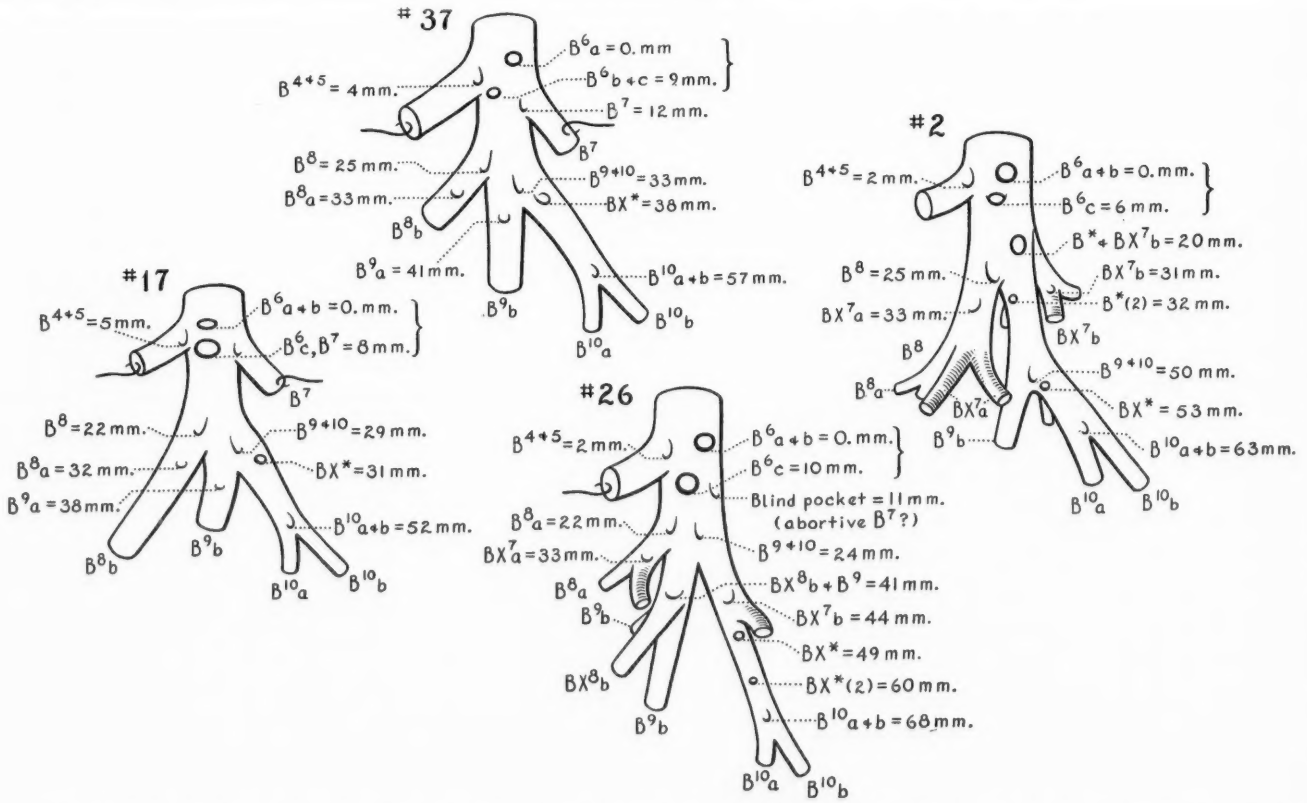


Fig. 1.—Sketches of the bronchial trees of four anomalous right lower lobes. Each has two orifices for the bronchi of the superior segment (B^6). The trees are drawn with B^7 retracted to the right and B^9 to the left. The keels of the bronchi are shown as if seen through transparent walls. Specimen #17 belongs to bronchial Type I (cf. Plate 4), #37 to Type III, and #26 and #2 to Type IV. (For arterial patterns of #17 and #26, see Fig. 2.) B^{4+5}, middle lobe bronchus; B^6a, b, c, medial, superior and lateral rami of superior segment; B^7, medial basal bronchus; BX^7a, BX^7b, displaced anterior and medial rami of B^7; B^8a, B^8b, lateral and basal rami of anterior basal bronchus; B^9a, B^9b, lateral and basal rami of lateral basal bronchus; $B^{10}a$, $B^{10}b$, laterobasal and mediobasal rami of posterior basal bronchus; B^*, subsuperior bronchus; BX^*, accessory subsuperior bronchus.

filled with gelatine it is believed that this deep stem was often missed. In any event, this branch *(B^6a2)* is the largest component of the medial ramus (cf. D, Plate 2, and "Lateral Surface," Plate 3). It swings around horizontally beneath the paravertebral surface in a plane just above the basal segments and extends to the posterior sector of the lobe and sometimes beyond that. Occasionally, it may arise separately (see bronchus indicated by a double dagger in Smith and Boyden's Plate 1).

The third observation deals with an increase in the number of cases of double origin of B^6. Four specimens were found (Fig. 1). In each case the opening of the middle lobe bronchus *(B^{4+5})* was located on a level between the two orifices of B^6. The distance between the orifices of B^6 ranged from 6 to 10 millimeters. Curiously enough Specimens #2 and #26 each had three independent arteries to the segment, while #17 and #37 had just two (Fig. 2).

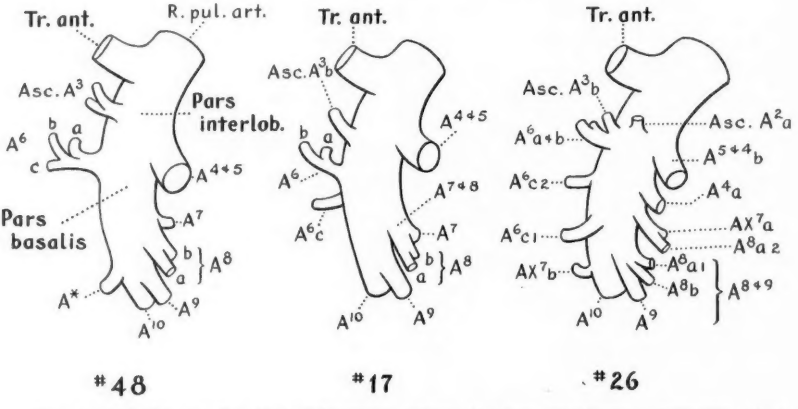


Fig. 2.—Sketches of the right pulmonary artery of three specimens. Specimen 48 is a representative of the prevailing bronchial Type I (cf. Plate 4). Its arterial pattern (except for A^6) is also of the prevailing type. #17 is normal except for two arteries to superior segment. #26 is atypical, having three arteries to the superior segment, displaced medial basal arteries (AX^7a, AX^7b) and two arteries to the anterior basal segment, one of which is associated with AX^7a, the other with A^2. *Tr. ant.*, truncus anterior to mediastinal surface of upper lobe; *Pars interlob.*, pars interlobaris, that portion of the pulmonary artery which appears in the depths of the interlobar fissure; *Asc.* A^2a, ascending artery to posterior ramus of anterior segment; *Asc.* A^3, ascending artery to interlobar side of the posterior segment of the upper lobe; A^{4+5}, middle lobe artery; A^6, artery of superior segment; *Pars basalis*, basal artery to segments 7 to 10; A^7, artery to medial basal segment; A^2, artery to anterior basal segment; A^9, A^{10}, arteries to lateral basal and posterior basal segments.

It is now possible to discuss the venous channels. It would simplify matters if one could say that the branching of V^6 is like that of the specimen shown in Plates 2 and 3, where three tributaries of approximately equal size *(V^6a, V^6b,* and *V^6c)*, lying on the anticlockwise side of their corresponding bronchi, unite to form V^6. However, the prevailing pattern (60 per cent) seems to be that of a single large stem formed from V^6b and V^6c. In this group V^6a is represented by one or more small branches entering the main trunk of V^6. Such an arrangement is shown in Plate 7. (The three small veins at the base of the trunk are subpleural, perhaps bronchial, veins.)

In the remaining 40 per cent of specimens, the tributaries of V^6 tend to have a shrublike arrangement and are too complicated to classify. However, it is important to note that some of the larger veins tend to be located between the superior and the basal segments.

Again, if one were to follow Overholt's principles of resection, A^6 would be ligated first (Fig. A, Plate 2), then B^6. This would expose the main stem of V^6. Subsequently, the plane of separation between the superior and basal segments would be developed along the line of the intersegmental branches of V^6a, V^6b and V^6c (Fig. D, Plate 2). In a third of the specimens, however, the plane between the two is oblique instead of horizontal (Smith and Boyden). Frequently, also, it is undulating, with the subsuperior zone protruding into the superior segment (Fig. 41 C, Plate 6).* In this connection, it should be noted that transverse fissures do not necessarily demarcate the intersegmental plane. In Plate 6 such a fissure separates the subsuperior zone *(BX*)* from the lateral basal segment *(B^9)* rather than from the superior segment *(B^6)*. In the same figure (#41 C), a vertical fissure descending toward the diaphragm separates two branches of B^9b, although higher up it coincides with the intersegmental plane between the anterior basal *(B^8a)* and lateral basal segment *(B^9a)*.

ANOMALIES OF THE INFERIOR PULMONARY VEIN

In the fifty specimens described in this article (and the 150 of the right lung dissected before that), no such gross anomaly as the drainage of the pulmonary veins into the systemic circulation has been encountered. (Brody records 68 such cases.) However, there were aberrant types of drainage into the inferior pulmonary vein in 22 per cent of the 50 specimens (Table III).

The common mode of formation of the inferior pulmonary (78 per cent) is by union of the superior vein *(V^6)* and the common basal vein. At the other extreme, is the pattern shown at *C* in the upper right corner of Plate 6. Here three vessels from the right lung join to form a common pulmonary vein. Healey and Gibbon found that in 3 per cent of 184 specimens the right lungs drained into a common right pulmonary vein. On the left side the figure rose to 25 per cent. The specimen in Plate 6 somewhat resembles three other cases by Healey and Gibbon (and two by Brantigan) in which a vein from each lobe of the right lung entered the left atrium; but in our specimen the middle vein drains not only a portion of the middle lobe but a portion of the superior segment and much of the lower lobe. Therefore, in intrapericardial ligation of veins, it would not be safe to assume that the three veins drain the respective lobes.

The second mode of formation of the inferior pulmonary vein is by union of three major veins (Table III). The simplest type is shown in Plate 7. The third mode is that in which four major veins are confluent.

These multiple types are due mostly to the downward displacement of veins not usually draining into the inferior pulmonary. Thus in 10 per cent the inferior pulmonary receives veins from the middle lobe (Plates 5 and 6; see also

*Tobin and Zariguicy (1950) have also stressed this fact.

Boyden and Hamre, Plate 5, specimen #38); in another 4 per cent, it receives the posterior vein of the right upper lobe *(V^3)*. This descends behind the root of the lung (see Boyden and Scannell, Plate 7, specimen #26).

TABLE III. MODE OF FORMATION OF RIGHT INFERIOR PULMONARY VEIN

I. Formed by union of *two* major veins			86%
1. Superior (V^6) and common basal vein		78%	
2. Miscellaneous combinations		8%	
V^6 + VX^3 and common basal**	2%		
V^6 + V^* and common basal (Plate 2)	2%		
V^6a, b and common basal (V^6c from sup. basal)†	2%		
V^4 + sup. basal (with VX^6c) and inf. bas. + V^6a, b (Plate 6, #5)*†‡	2%		
II. Formed by union of *three* major veins			10%
V^6; sup. basal; inf. basal	4%		
V^6; V^4 or V^{4+5}; common basal‡	4%		
V^6; VX^3; common basal**	2%		
III. Formed by union of *four* major veins			4%
V^4; V^6; sup. basal; inf. basal (Plate 6)‡	2%		
V^{4+5}; V^6; sup. basal; inf. basal (Plate 5)‡	2%		

*These two drain into a common pulmonary vein (See #5C, Plate 6).
**Inferior pulmonary receives posterior vein (V^3) of upper lobe (4 per cent).
†Inferior pulmonary receives two veins from superior segment (4 per cent).
‡Inferior pulmonary receives veins from middle lobe (10 per cent).

THE GENERAL ARTERIAL PATTERN

If one looks into the interlobar fissure of an undissected right lung, one sees the interlobar portion of the right pulmonary artery (pars interlobaris) at the bottom of the fissure betwen the upper and middle lobes, partly covered by the posterior vein of the upper lobe (compare Boyden 1945, Fig. 3). If the middle lobe be reflected upward, the pars interlobaris may then be seen lying lateral and superficial to the corresponding stem bronchus (the bronchus intermedius of Ewart), Figs. E and A, Plate 2. From this site, the artery curves backward along the posterolateral aspect of the lower lobe bronchus. Accordingly, in a dissection the artery is best seen in a lateral view (Plate 3). In a postero-anterior film it has much the same slant, appearing as branched markings along the right border of the heart, as originally demonstrated in angiograms by Carvalho (1936).*

The *pars interlobaris* (Fig. 2), that part of the right pulmonary artery which lies between the *truncus anterior* (the principal artery to the upper lobe) and the middle lobe artery *(A^{4+5})*, gives rise to one or more ascending arteries of the upper lobe in approximately three-fourths of the lungs (Boyden and Scannell). In 52 per cent of lungs there are two middle lobe arteries (Boyden and Hamre). Liard found three in 4 per cent of specimens. The lower lobe artery is a continuation of the pars interlobaris. Almost immediately it gives off the artery to the superior segment (A^6) and then continues as the *pars basalis,* the trunk which supplies the basal segments (Fig. 2, #48). If there are no complications, A^6 is given off slightly below the level of the middle lobe

*We are indebted to Prof. Ernest Lachman for the privilege of seeing these beautiful plates.

artery. If there are two middle lobe arteries A^6 lies at an intermediate level, i.e., in a plane drawn between the two. If there are two or more arteries to the superior segment, each may lie just below the corresponding middle lobe artery (Boyden and Hamre, Fig. 3); if there are three, the highest may lie above the middle lobe arteries, and the lowest below them (Fig. 2, #26).

THE ARTERIES OF THE SUPERIOR SEGMENT (A^6)

As summarized in Table IV, a single artery supplies the segment in 80 per cent of specimens. The prevailing pattern (50 per cent) is a bifurcation into a lateral ramus (A^6c) and into combined medial and superior rami ($A^6a + A^6b$, Plate 2). This, it will be recalled, is also the prevailing pattern of the bronchi (72 per cent, Table II), but an analysis of all specimens indicates that a given arterial pattern reproduces the bronchial pattern in only 56 per cent. In other words the arterial rami cross the subsegmental planes in 44 per cent of specimens. Sometimes the arteries trifurcate, or are branched like a shrub or merely have another type of bifurcation.

In 16 per cent there are two separate arteries (cf. #17, Fig. 2) and in 4 per cent three separate arteries (cf. #26, Fig. 2). In sum, more than one artery is to be looked for in one-fifth of the lobes. Preoperatively, these could not be predicted except in those cases in which two orifices for B^6 have been demonstrated by bronchography or at bronchoscopy. (See discussion following Table II.)

Another impediment to the resection of this segment is the fact that in 10 per cent of specimens A^6 carries a displaced ascending artery to the upper lobe. Almost always this supplies the posterior segment (cf. A^3b, Fig. 2, #26).

In concluding this section, attention should be called again to the importance of the interlobar part of the right pulmonary artery in segmental resection and lobectomy (cf. Lindskog, Liebow and Hales; Boyden and Hamre).

TABLE IV. ARTERIAL SUPPLY OF SUPERIOR SEGMENT

I. Segment supplied by *single* artery bifurcating into:			80%
1. Simple types		64%	
1) A^6c and $A^6a + b$ (Plate 2)	50%		
2) A^6a and $A^6b + c$ (Fig. 2, #48)	8%		
3) A^6b and $A^6a + c$	6%		
2. Complex types (with displaced branches)*		16%	
II. Segment supplied by *two* separate arteries (Fig. 2, #17)			16%
1. Simple types (A^6a or A^6c separate from the rest)		8%	
2. Complex types		8%	
III. Segment supplied by *three* separate arteries (Fig. 2, #26) A^6c split, or A^6b split			4%

*For example, $A^6b + a1$ and $A^6c + a2$.

THE ARTERIES TO THE MEDIAL AND ANTERIOR BASAL SEGMENTS (A^7, A^8)

In seeking a method by which the branches of the basal portion of the pulmonary artery may be classified it has been necessary to eliminate the "dorsal" rami (subsuperior arteries), since they are as variable in origin as the corresponding bronchi (B^*, BX^*). When these are ignored, the distribu-

tion of arteries to the medial basal and anterior basal segments *(B^7* and *B^8)* seems to be correlated with the bronchial patterns shown in Plate 4. In the 56 per cent of specimens which makes up Types I and II (the normal pattern) the first anterior branch of the basal artery is either the medial basal (38 per cent; cf. #48, Fig. 2) or *A^{7+8}* (18 per cent; cf. #17, Fig. 2).* In the remaining 44 per cent of specimens (Types III and IV, in which *B^7* either surrounds the basal veins or is absent as such) all but three of the specimens (6 per cent) are characterized by a displacement of the medial ramus *(A^7b)*. That is, the latter shifts posteriorly, either to arise from the basal artery or to become an accessory branch of *A^**, *AX^** or *A^{10} (AX^7b,* Plate 5, #24). Meanwhile *A^7a* remains in its usual anterior position *(A^7a,* Plate 5, #1) or shifts laterally to become an accessory branch of *A^8* or *A^9 (AX^7a,* Plate 5, #24).

Another striking feature is the variability of the artery to the anterior basal segment *(A^8)*. Smith and Boyden showed that *B^8* was the most constant of the basal bronchi of the right lung, and this has been verified in our series; for in 94 per cent of specimens it arises as a single branch of the stem bronchus.† Yet the corresponding artery *(A^8)* arises as a single branch of the basal artery in only 48 per cent of specimens. In another 16 per cent (12 and 4) it is associated with either *A^7* or *A^7a* (cf. Plate 5, #1); in another 12 per cent it is associated with *A^9* (cf. Plate 5, #24). In the remaining 24 per cent it is split, arising as two separate arteries from the basal trunk (Plate 3) or as two separate arteries associated with portions of *A^7* or *A^9*, or with the whole of *A^9*.

Perhaps the variability of *A^8* may be explained by the fact that it lies next to a variable segment, but this does not explain the numerous variations encountered in other basal arteries (vide infra). In any event the importance of these arterial anomalies of *A^8* are obvious since hitherto the anterior basal segment has been considered one of the easily resectable segments.

THE ARTERIES TO THE LATERAL AND POSTERIOR BASAL SEGMENTS *(B^9, B^{10})*

The basal portion of the right pulmonary bronchus may be considered to terminate in a bifurcation—namely, in the lateral basal and posterior basal segmental bronchi. In the fifty specimens studied by us, *B^9* was absent as such—that is it developed as an accessory branch of *B^8*—in only 4 per cent. (Smith and Boyden found it in 8 per cent.) Similarly, in our study there was only 10 per cent of specimens in which *B^9b* arose from *B^{10}*, or *$B^{10}a$* from *B^9b*.‡

Accordingly, one would expect that with relatively so few variations in the forking of the bronchi (14 per cent) there would be correspondingly few variations in the forking of the arteries. Strangely enough, however, the basal artery terminates normally by bifurcating into segmental arteries *A^9* and *A^{10}* in only 44 per cent of specimens (cf. Plate 3).

*In three specimens there were minor variations: in one, the basal artery trifurcated into A^7, A^8, A^{9+10}; in the other two, A^8a (or a part of it, Fig. 2, #26) branched off lower down (Plate 3).

†In the remaining specimens (6%), B^8a and B^8b had separate origins of varying sorts (cf. Fig. 1, #26).

‡There are other variations which do not affect the arrangement of arteries, such as an absence of B^8a in 16% and the presence of a high paravertebral branch of B^{10} in 14%.

In the remainder many types of variations occur. Thus in 18 per cent A^9 is split, its two rami arising separately from the basal artery or in common with branches to other segments. In 30 per cent, A^9 is atypically associated with A^8, A^{7+8}, A^* or AX^*; and in 8 per cent the branches of A^9 and A^{10} are mixed.

THE ARTERIES OF THE SUBSUPERIOR ZONES (A^* AND AX^*)

A summary of the occurrence and origin of the subsuperior and accessory subsuperior bronchi in 100 specimens (from our data and those of Smith and Boyden) is presented in Table V. It will be recalled that B^* corresponds to the second dorsal ramus of Aeby, BX^* to the third and B^6 to the first. (See succession of dorsal rami ending in $D4$ in Plate 7.)

Forty-two arteries to 36 subsuperior bronchi (B^*) occur in thirty of our fifty specimens. In exactly four-fifths of these the arteries arise on the dorsal side of the basal artery either high up (51 per cent), in the vicinity of A^7 or A^8 (Plate 6, #5A), or lower down (29 per cent), nearer the bifurcation of the basal artery into A^9 and A^{10} (Plate 5, #1). The remainder arise aberrantly from A^{10} (10 per cent) or A^9 (10 per cent). The larger number of arteries is due to the fact that the arterial rami which accompany a given subsuperior bronchus may arise from more than one site. Because of the deep posterior location and the variable origins of these arteries, resection of a subsuperior segment would seem to be almost impossible unless the superior segment (B^6) had been removed first.

TABLE V. SUBSUPERIOR BRONCHI (B^* AND BX^*)

I. Frequency of occurrence in 100 specimens			
1. Subsuperiors arise exclusively from level d_2 of stem bronchus as B^*			16%
2. Subsuperiors arise exclusively from level d_2 as BX^* from B^{10}			39%
3. Subsuperiors arise from both levels in same specimen			45%
			100
II. Detailed analysis			
1. Subsuperior represented by a single bronchus		43%	
(1.) Arising at d_2 level, as B^*	13%		
(2.) Arising at d_2 level, as BX^* (10)	30%		
2. Subsuperior represented by two bronchi		43%	
(1.) Two B^*	3%		
(2.) One B^* and one BX^* (10)	32%		
(3.) Two BX^* (10)	8%		
3. Subsuperior represented by three bronchi		13%	
(1.) Two B^* and one BX^*	8%		
(2.) One B^* and two BX^*	4%		
(3.) Three BX^*	1%		
4. Subsuperior represented by four bronchi			
(1.) Two B^* and two BX^* (10)		1%	
III. Origin of B^* (at d_2 level). (Items I:1 and I:3)			61%
1. One subsuperior		48%	
(1.) At level of B^7	10%		
(2.) Between B^7 and B^8	18%		
(3.) Between B^8 and B^9	20%		
2. Two subsuperiors		13%	
(1.) At level of B^7 and between B^8 and B^9	2%		
(2.) Between B^7 and B^8 and between B^8 and B^9	5%		
(3.) Between B^8 and B^9	6%		

Fifty-eight arteries to 52 accessory subsuperior bronchi *(BX*)* occur in forty-one of our fifty specimens. Nearly seven-eighths of these (86 per cent) arise from A^{10}, 9 per cent from the lower part of the basal artery, and 5 per cent from A^{9}. Thus, as might be expected, the subsuperior arteries originate prevailingly from the upper half of the basal artery while the accessory subsuperiors arise chiefly from A^{10}.

Returning to the bronchi, only one correlation can be made between B^{*} and the four patterns presented in Plate 4, but this is very striking. In Type III, B^{*} is present in only 42 per cent of specimens of that group, whereas in Types I, II and IV the respective percentages are 59, 82, and 60. From these figures it would appear that when $B^{7}b$ lies on the deep side of the basal vein (Type III) there is the least room for subsuperiors to develop and when B^{7} lies most anterior (Type II) there is most room.

Another interesting comparison can be made between the position of the highest BX^{*} (the high posterior branch of B^{10}) and $B^{9}a$, the high posterior-lateral branch of B^{9}. The keel of each lies about a centimeter below the keel separating B^{9} from B^{10} (Fig. 1, #17 and #37), but that of BX^{*} is slightly higher, averaging 7.2 mm., with a range of from 2 to 15 mm. (thirty-eight specimens) while that of $B^{9}a$ averages 11 mm. with a range of from 7 to 14 mm. (thirty-nine specimens).*

CONCLUSIONS

Analysis of the segmental bronchi, veins, and arteries in fifty dissected specimens of the right lower lobe has led to the conclusion that the most important relation in the basal half of the lobe is that between the bronchi of the medial basal segment and the large venous trunks which enter the inferior pulmonary vein.

The inferior pulmonary is formed by a confluence of the vein from the superior segment *(V6)* and the common basal vein. The latter is formed by the union of superior and inferior basal veins that drain the basal segments. The upper basal comes largely from segments 8 and 9, the lower basal chiefly from segments 9 and 10.

The mode of formation of the inferior pulmonary vein is anomalous in 22 per cent of specimens. It may be formed by three or four trunks and receive major tributaries from the middle and even the superior lobe.

Four types of the medial basal segment *(B^{7})* are recognized: (I), the prevailing pattern, that in which the segmental bronchus *(B^{7})* is distributed to both anterior and paravertebral surfaces, and passes superficial to the basal veins (34 per cent); (II) a slight modification of the prevailing pattern in which the distribution of B^{7} is limited to the anterior (and diaphragmatic) surfaces (22 per cent); (III), a major variation in which B^{7} divides around the common basal vein (24 per cent); (IV), a major variation in which B^{7} is absent as such (20 per cent), $B^{7}a$ arising as an accessory branch of the anterior basal bronchus *(B^{8})*, and $B^{7}b$ arising as an accessory branch of the subsuperior *(B^{*})* or the posterior basal bronchus *(B^{10})*.

Resection of Type III is considered to be hazardous, and criteria are presented by which it can be recognized preoperatively. Resection of Types I and II are feasible, and a description is presented of the intersegmental veins along which the segment may be resected.

Similarly, attention is called to the intersegmental veins which separate the superior *(B^{6})* from the basal segments *(B^{7-10})*. The arteries to the superior segment are multiple in one-fifth of specimens. In 8 per cent the bronchi of the superior segment arise from two separate orifices 6 to 10 mm. apart; thus the lower B^{6} might be confused with a high subsuperior bronchus *(B^{*})*.

*In additon to the thirty-nine there are three specimens in which $B^{9}a$ arises atypically at the orifice of B^{9}. In the remaining eight it is absent.

The basal portion (pars basalis) of the inferior pulmonary artery lies on the posterolateral side of the stem bronchus. In 44 per cent, it terminates by forking into A^9 and A^{10}, the arteries of the lateral basal and posterior basal segments. In the remainder, the lower arteries arise irregularly. The first anterior branch of the basal artery is A^7 (or A^{7+8}) in Types I and II (56 per cent) but in Types III and IV (44 per cent) A^7b is nearly always posterior in origin. A^8 arises as a single branch of the basal artery in only 48 per cent. In the remainder it is split in origin (24 per cent) or arises in conjunction with A^7 or A^9 (28 per cent). The situation is further complicated by the variable origin of multiple arteries to the subsuperior zones (100 in fifty specimens).

REFERENCES

Adachi, Buntaro: Anatomie der Japaner. II. Das Venensystem der Japaner, Kyoto, Kenkyusha Press, 1933.

Boyden, Edward A.: The Intrahilar and Related Segmental Anatomy of the Lung, Surgery **18:** 706-731, 1945.

Boyden, Edward A.: A Synthesis of the Prevailing Patterns of the Bronchopulmonary Segments in the Light of Their Variations, Dis. Chest **15:** 657-669, 1949.

Boyden, E. A., and Hamre, C. J.: An Analysis of Variations in the Bronchovascular Patterns of the Middle Lobe in Fifty Dissected and Twenty Injected Lungs, J. THORACIC SURG. **21:** 172-188, 1951.

Boyden, Edward A., and J. Gordon Scannell: An Analysis of Variations in the Bronchovascular Pattern of the Right Upper Lobe of Fifty Lungs, Am. J. Anat. **82:** 27-74, 1948.

Brantigan, O. C.: Anomalies of the Pulmonary Veins—Their Surgical Significance, Surg., Gynec. & Obst. **84:** 653-658, 1947.

Brock, R. C.: The Anatomy of the Bronchial Tree, With Special Reference to the Surgery of Lung Abscess, London, 1946, Oxford University Press.

Brock, R. C.: Post-tuberculous Broncho-stenosis and Bronchiectasis of the Middle Lobe, Thorax **5:** 5-39, 1950.

Brody, Henry: Drainage of the Pulmonary Veins into the Right Side of the Heart, Arch. Path. **33:** 221-240, 1942.

Carvalho, Lopo de: Les Aspects Radiologiques Du Hile et Leur Interpretation. X. Conferencia da Vnião Internacional Contra A Tuberculose. Lisboa, 1936. (34 pp., 12 pl.)

Dévé, F.: Les Lobes Surnuméraires du Poumon, Bull. et Mém. Soc. Anat. **75:** 341-367, 1900.

Healey, John E., Jr., and Gibbon, John M.: Intrapericardial Anatomy in Relation to Pneumonectomy for Pulmonary Carcinoma, J. THORACIC SURG. **19:** 864-874, 1950.

Herrnheiser, Gustav, and Kubat, Anton: Systematische Anatomie der Lungengefässe, Ztschr. f. Anat. u. Entweklngsgesch. **105:** 570-653, 1936.

Liard, A. Ruiz: Contribúcion al Estudio de los Pediculos Pulmonares. II Pediculo Lobar Medio Derecho, Anales Facultad de Medicina Montevideo **35:** 41-74, 1950.

Lindskog, G. E., Liebow, A. A., and Hales, M. R.: Bilobectomy-Surgical and Anatomic Considerations in Resection of Right Middle and Lower Lobes Through the Intermediate Bronchus, J. THORACIC SURG. **18:** 616-629, 1949.

Melnikoff, Alexander: Die chirurgische Anatomie des intrapulmonen Gefässe und der Respirationswege, Arch. f. klin. Chir. **124:** 460-510, 1922.

Overholt, Richard, H., Woods, Francis M., and Ramsay, Beatty H.: Segmental Pulmonary Resection, J. THORACIC SURG. **19:** 207-220, 1950.

Scannell, J. Gordon: An Anatomic Approach to Segmental Resection, J. THORACIC SURG. **18:** 64-74, 1949.

Smith, Franklin R., and Boyden, Edward A.: An Analysis of Variations of the Segmental Bronchi of the Right Lower Lobe of Fifty Injected Lungs, J. THORACIC SURG. **18:** 195-215, 1949.

Tobin, Charles E., and Zariquiey, Manuel O.: Bronchopulmonary Segments and Blood Supply of the Human Lung. A radiographic and Anatomic Study, Med. Radiog. & Photog. **26:** 38-45, 84, 1950.

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LOBECTOMY FOR FUNGOUS ABSCESS OF THE LUNG: EFFECT OF PENICILLIN

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THERE is a good deal of evidence to show that the administration of penicillin encourages fungous infections, and it is probably for this reason that these infections are on the increase. The following case is of interest because:

(1) A huge pulmonary abscess was treated with penicillin which eradicated all organisms except a fungus.

(2) Heroic doses of penicillin nevertheless kept the clinical symptoms under control while the patient was being prepared for surgery.

(3) The patient survived lobectomy in spite of his age and desperate condition.

CASE REPORT

The patient was a 72-year-old, American-born grocer who was first observed by one of us (M.M.) in October, 1949. Presenting symptoms were cough, hemoptysis, weakness, fever, and nosebleeds. He had been in good health until 1947 when a chest film showed a density in the right apex. He started going downhill, but he continued active until September, 1949, when the above-mentioned symptoms of lung abscess developed. Sputum was mucopurulent, occasionally bloody. Fever went as high as 102° F.

Physical examination showed that the man was critically ill. He was dyspneic, cyanotic, toxic, and emaciated. His blood pressure was 122/74; pulse was 80. There was a maximal precordial impulse in the fifth intercostal space at the midclavicular line. A low, blowing, musical, systolic murmur replaced all heart sounds, and was transmitted over the entire upper chest. Rhythm was regular. Showers of râles were present at the right apex. Prostate was enlarged, grade I. Diffuse, patchy, scaly eruption over the sternum was noted.

A chest film revealed a giant cavity in the right upper lobe, measuring 4.7 cm. by 7 cm. (Fig. 1). There was no fluid level, but the lower three-fourths of the cavity contained a "cotton ball" density. A lateral view showed the cavity to be occupying the posterior segment of the upper lobe. The interlobar fissure was shifted upward, and the trachea was pulled to the right. The remainder of the lung showed fibrosis and emphysema. The heart was normal in size. An electrocardiogram revealed T-wave and ST-segment changes indicative of myocardial disease.

Laboratory work showed hypochromic anemia, hypoproteinemia, and mild azotemia. There was a persistent eosinophilia varying from 5 to 21 per cent. Sputum cultures at first showed a monilia-like fungus. Later they became sterile. Bronchoscopy showed purulent material coming from the right upper lobe bronchus. This material failed to show a fungus on culture. A needle biopsy somewhat later in the course of the illness showed a great abundance of septate mycelium in necrotic lung tissue. Morphologically it resembled Aspergillus; culturally it was sterile. Intradermal tests for cococcidioidin and histoplasmin were negative in twenty minutes and forty-eight hours. Oidiomycin showed a two plus reaction in twenty minutes with no delayed reaction. An autogenous vaccine of the monilia-like fungus from the initial sputum culture showed a two plus reaction on intradermal tests in twenty minutes.

Received for publication March 15, 1951.

Course.—Therapy was directed towards combating the pulmonary infection and overcoming the toxemia. The patient was treated with intravenous amino acids, blood transfusions, crude liver extract, and vitamins. Digitalis was given from the start. Parenteral penicillin was started with an initial dose of 1 million units a day. The temperature which at first ranged as high as 104° F. returned to normal, and the symptoms improved. On November 11 the penicillin was cut to 240,000 units a day. In a few hours the patient showed an increase in toxicity with a rise in temperature. Because of this, the penicillin was increased to 500,000 units per day and then gradually to 1 million, 3 million, 8 million, and finally 20 million units a day. The cavity shrank noticeably (Fig. 2). Three times an attempt was made to reduce the dose, but each time the patient reacted with signs of increased toxicity. On two of these occasions he suffered massive hemoptysis. Aureomycin, chloromycetin, gantrisin, dihydrostreptomycin, and iodides were all tried but without apparent benefit.

The patient now developed urinary obstruction. Because his condition was still too poor for surgery, he was placed on catheter drainage, and after several weeks he had a transurethral prostatectomy. He recovered. A few days later the needle biopsy of the lung was repeated. The findings were the same as at first.

Fig. 1.—Appearance of the lesion when the patient was first seen.

Surgery for the pulmonary lesion was undertaken on March 3, 1950 (C.L.G.). At thoracotomy the right upper lobe was found densely adherent to the parietal pleura. The posterior segment of the upper lobe was heavily infiltrated, and this process also involved the superior segment of the lower lobe. Because of the extent of the disease, and because carcinoma was still not entirely ruled out, a lobectomy was decided upon. The hilum was surprisingly free. The branches of the pulmonary artery and of the superior pulmonary vein were individually ligated and divided. The bronchial stump was closed with 00 silk. A small amount of the superior segment of the lower lobe was also removed. The wound was closed, and two catheters were inserted into the chest, one in the posterior axillary line, and one in the midclavicular line. Through these catheters penicillin and streptomycin were inserted into the pleural cavity during the first two postoperative days.

The patient did poorly at first because of severe bronchial asthma and cerebral anoxia. He was confused, agitated, and disoriented. These symptoms gradually subsided. The lung

expanded well, except for a small pocket at the apex which has persisted to date. Vital capacity has remained practically unchanged. The patient went home on the twelfth postoperative day and has remained well (Fig. 3).

Examination of the operative specimen showed a chronic lung abscess 5 cm. in diameter, draining into the posterior division of the right upper lobe bronchus. It was filled with a reddish-black, necrotic slough. Microscopic examination showed that the walls were covered with the same type of fungus as had been noted in the needle biopsies. Cultures were again sterile.

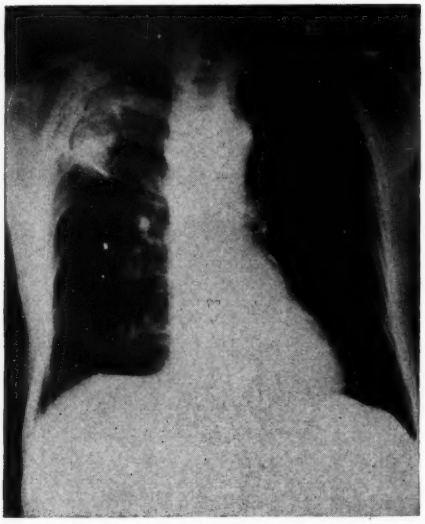

Fig. 2.—After three months of penicillin therapy.

COMMENT

In summary, this was an elderly patient who developed a pulmonary lesion of undetermined nature in 1947. Two years later this lesion rapidly broke down and formed a giant abscess which was partially controlled with tremendous doses of penicillin. Cultures were negative except at the very beginning (before penicillin was started). Two needle biopsies and the operative specimen showed a fungus, either Candida or Aspergillus. After a stormy preoperative course the patient came to lobectomy. He survived, and is now as well as ever.

There are several interesting features in this case. In the first place, what was the role of penicillin? Did it enhance the growth of the fungus, or did it simply suppress the normal competitors? Secondly, why did penicillin produce clinical improvement and shrinkage of the cavity? Thirdly, this patient illustrates that lobectomy for lung abscess is not necessarily contraindicated in the presence of old age, general debility, advanced arteriosclerosis, and severe cardiac damage.

The effect of penicillin on fungi has been made the subject of several investigations and a number of case reports. As early as 1942 Hobby, Meyer, and Chaffee[1] tested various strains of fungi for penicillin-sensitivity in the test

tube and found that all were penicillin-resistant with the exception of Cryptococcus hominis. In 1944 Keeney, Ajello, and Lankford[2] found that *Candida albicans, Cryptococcus neoformans, Histoplasma capsulatum,* and *Coccidioides immitis* were not only unaffected, but actually stimulated by penicillin in the test tube. In 1946 Fleming and Queen[3] repeated these experiments with nineteen different strains of fungi. Their conclusions were that all were penicillin-resistant, with the exception of Actinomyces (there is also good clinical evidence that actinomycosis is favorably influenced by penicillin[4]). In 1947 Mattman[5] reported that the common fungi were consistently resistant to penicillin and to streptomycin in laboratory tests. In 1949 Foley and Winter[6] noted that throat

Fig. 3.—Postoperative appearance of the chest.

cultures from patients treated with penicillin for diphtheria and scarlet fever often showed an abnormal preponderance of fungi. They then injected various fungi into chick embryos and observed that the mortality was greatly increased by the addition of penicillin. They thought that penicillin had a direct stimulating effect on the fungus, rather than a suppressing effect on competitive organisms.

Besides these experimental observations, there are several case reports suggesting that penicillin makes the patient susceptible to fungi. In 1946 Geiger, Weimer, Axilrod, and Durlacher[7] described a patient who developed a fatal *Candida albicans* endocarditis and meningitis during intensive penicillin therapy for subacute bacterial endocarditis. In 1950 Grekin, Cawley, and Zheutlin[8] reported fatal pulmonary aspergillosis in a patient who was under penicillin treatment for influenza. In the same year Zimmerman[9] reported three cases of Candida and Aspergillus endocarditis, all of whom had been under treatment with penicillin and various antibiotics. He felt that there was an etiological connection and quoted as yet unpublished work by Farber pointing in the same

direction. In 1951 Woods, Manning, and Patterson[10] reported twenty-five patients who developed monilial infections while under treatment with penicillin, aureomycin, and chloramphenicol. Two of these patients had pulmonary lesions. These authors believe that the patient is made susceptible by the changes which the antibiotics produce in the normal intestinal and bronchial flora. Other authors, such as Schnurman[11] and Keeney,[2] have seen acute exacerbations of mycotic skin infections under parenteral penicillin therapy for unrelated infections. This is believed to be an allergic manifestation. The only author to report a favorable effect of penicillin on pulmonary moniliasis is Zussman,[12] but his patient received so little of the drug (a total of 1.5 million units) that it is questionable whether there was any causal relationship.

With all this evidence at hand, it seems probable that our patient had a mixed infection in which the fungi eventually became the predominating organisms. Fungi are normal inhabitants of the mouth in many people. The significant observation here is that it was possible to keep the clinical symptoms under control and even produce some shrinkage of the cavity, not by the discontinuance of the penicillin (as others have suggested) but rather by an increase in the dose to the heroic levels of 20 million units a day. Several attempts to reduce this dose led to a clinical relapse. This is difficult to explain on the basis of the known penicillin-resistance of fungi. Possibly the suppression of all other organisms had something to do with it. At any rate, it is a fact well worth recording because it was this temporary control of the infection that made operation eventually possible.

We were never able to identify the fungus in this case. Morphologically it looked like Candida or Aspergillus because of the profuse mycelium, but the morphologic characteristics of fungi are notoriously variable, and it is necessary to have cultural evidence for a positive diagnosis. However, even had we been successful in culturing this organism, the information would not have had any important bearing on the treatment. Clinically these infections are a single entity, and any variations that occur are based on fortuitous fluctuations in virulence rather than on specificity of strains.

The preferred treatment of chronic lung abscess has undergone a noticeable change in the last ten years. Whereas simple drainage used to be the treatment of choice, resection is now carried out more and more frequently. A simple drainage would never have cured this patient. There was too much destruction of pulmonary tissue. Also, there is always the question of a hidden cancer. In recent years Shaw and Paulson,[13] as well as Kent and Ashburn[14] have stressed the desirability of resection. They feel that a great many patients continue to have symptoms of residual bronchiectasis when an abscess is merely drained. The oldest patient in the series of Shaw and Paulson was 54 years of age. The oldest patient in the series of Kent and Ashburn was 64 years of age. Our patient was 72 years, but he is not the oldest patient in the literature to survive lobectomy for abscess. Rosenblatt, Aufses, and Lisa[15] have reported a successful right middle lobectomy in a patient 82 years old!

Nevertheless, the result achieved here was most gratifying. The patient was debilitated, toxic, senile, and extremely ill. He was suffering from an over-

whelming infection, and nearly died of massive hemoptysis on several occasions. In addition, he had generalized arteriosclerosis, advanced pulmonary emphysema, severe cardiac damage, and long-standing prostatism. That such a patient could survive was due chiefly to the careful preoperative preparation, meticulous attention to detail during the operation, and proper supportive measures afterwards. Penicillin played an important role.

SUMMARY

A man 72 years of age who was severely ill with a huge pulmonary abscess that showed only fungus organisms responded to massive doses of penicillin and survived a right upper lobectomy.

REFERENCES

1. Hobby, G. L., Meyer, K., and Chaffee, E.: Activity of Penicillin in Vitro, Proc. Soc. Exper. Biol. & Med. **50:** 277, 1942.
2. Kenney, E. L., Ajello, L., and Lankford, E.: Studies on Common Pathogenic Fungi and on Actinomyces Bovis, Bull. Johns Hopkins Hosp. **75:** 410, 1944.
3. Fleming, R. S., and Queen, F. B.: Penicillin Resistance of Fungi, Am. J. Clin. Path. **16:** 66, 1946.
4. Putman, H. C., Dockerty, M. B., and Waugh, J. M.: Abdominal Actinomycosis, Surgery **28:** 781, 1950.
5. Mattman, L. H.: Penicillin and Streptomycin Sensitivity of Miscellaneous Bacteria and Fungi, Am. J. Med. Tech. **13:** 159, 1947.
6. Foley, G. E., and Winter, W. D.: Increased Mortality Following Penicillin Therapy of Chick Embryos Infected With Candida Albicans, Var. Stellatoidea, J. Infect. Dis. **85:** 268, 1949.
7. Geiger, A. J., Weimer, H. A., Axilrod, H. D., and Durlacher, S. H.: Mycotic Endocarditis and Meningitis, Yale J. Biol. & Med. **18:** 259, 1946.
8. Grekin, R. H., Cawley, E. P., and Zheutlin, B.: Generalized Aspergillosis, Arch. Path. **49:** 387, 1950.
9. Zimmerman, L. E.: Candida and Aspergillus Endocarditis, Arch. Path. **50:** 591, 1950.
10. Woods, J. W., Manning, I. H., and Patterson, C. N.: Monilial Infections Complicating Therapeutic Use of Antibiotics, J. A. M. A. **145:** 207, 1951.
11. Schnurman, A. G.: Five Cases of Latent Trichophyton Infection Activated by the Use of Penicillin, Virginia M. Monthly **73:** 281, 1946.
12. Zussman, B. M.: Pulmonary Moniliasis Treated With Penicillin, J. Tennessee M. A. **40:** 258, 1947.
13. Shaw, R. R., and Paulson, D. L.: Pulmonary Resection for Chronic Abscess of the Lung, J. THORACIC SURG. **17:** 514, 1948.
14. Kent, E. M., and Ashburn, F. S.: Pulmonary Resection for Chronic Lung Abscesses, J. THORACIC SURG. **17:** 523, 1948.
15. Rosenblatt, M. B., Aufses, A., and Lisa, J. R.: Lobectomy in a Patient Eighty-Two Years of Age, J. A. M. A. **143:** 552, 1950.

DOUBLE AORTIC ARCH

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IN THE year 1794 Bayford applied the name of *dysphagia lusoria* to a morbid condition characterized by difficulties in swallowing and the presence of an abnormally located right subclavian artery, which passes posterior to the esophagus, causing difficult swallowing owing to its pressure. Since that date a number of anomalous developments of the aortic arch have been observed which may give rise to similar clinical symptoms. One of them is the double aortic arch, a rare irregularity; in this case the ascending aorta divides into two branches encircling the esophagus and trachea (very rarely the trachea only), uniting a little distance farther into the descending aorta.

In normal circumstances the arcus aortae develops from the left fourth branchial arch, the innominate artery and the right subclavian artery springing from the right fourth branchial arch, and the distal portion of this primitive right aortic arch disappears (Fig. 1). When development of these arteries is disturbed during the period between the third and the sixth embryonal weeks, the result may be the formation of a ring around the esophagus and trachea, consisting wholly or partly of blood vessels; this ring, if sufficiently narrow, may cause pressure on the organs within it. Sometimes the pulmonary artery and the arterial duct (both arising from the sixth branchial arch) are also involved in exerting this pressure. Examples of the latter possibility are mentioned by Gross and Ware[7] when they describe the different vascular anomalies that may be observed in this region.

According to Gross and Ware,[7] a double aortic arch in man may be observed in two shapes:

1. As a partly obliterated double aortic arch. Only one of its legs (the right one usually) is open and situated behind the esophagus; the other has become obliterated and is represented by a vestigial remnant or cord in front of the trachea. This irregularity is less common than the second.

2. As a completely open double aortic arch. The two arterial branches very rarely have the same diameter (Schall and Johnson[12]), in most cases one is wider than the other. We nearly always find a wide right arch, passing posterior to the esophagus and trachea, and becoming a descending aorta, not situated to the right of the vertebral column, yet slightly farther to the right than a normal descending aorta. The left arch is present in the shape of a slenderer arterial branch, anterior to the trachea and uniting farther toward the periphery with the right arch, so as to form the descending aorta. The great arterial vessels arising from the aortic arch, viz., innominate artery, left carotid artery, and left subclavian artery, may arise in different ways from

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Received for publication March 23, 1951.
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the left and right aortic arches. Among the forty cases examined by Blincoe[3] there were only two where the left (anterior) arch was the wider. Similar cases were described by Gross,[6] Gordon,[5] and Potts.[10]

There are some cases in medical literature, however, where the trachea only was encircled, the posterior arch passing between the trachea and esophagus (Malacarne,[8] Sagorsky,[11] and Welch[14]).

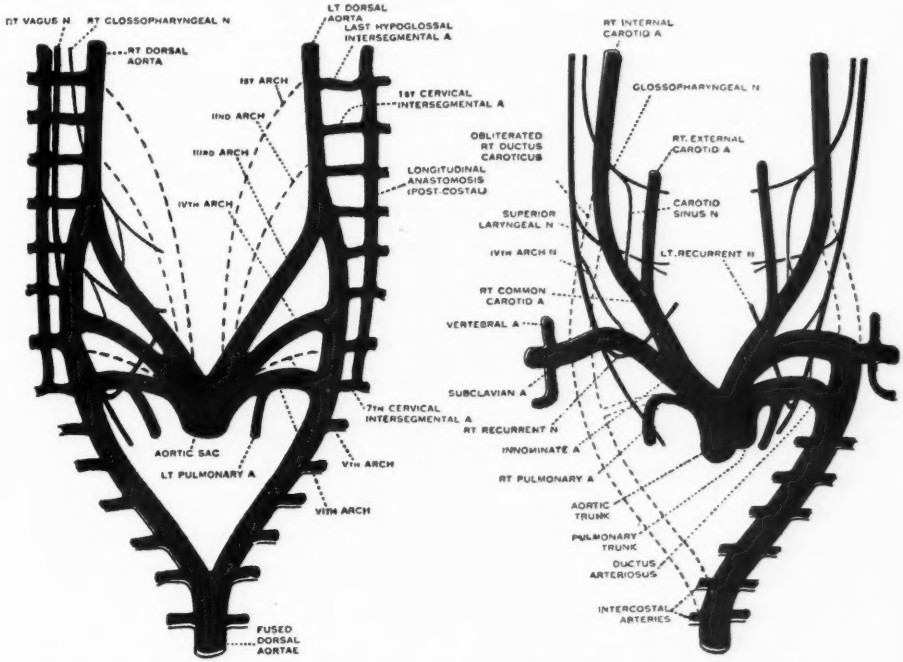

Fig. 1.—Normal development of aortic arch in man (from Hamilton, Boyd, and Mossman, *Human Embryology*).

It goes without saying that this vascular ring may compress and constrict the encircled anatomic organs. When this ring is sufficiently wide, this irregularity need not be attended with clinical symptoms; instances of this are mentioned in medical literature (Arkin[2]). Should the ring be narrow, however (and this seems to be the case in the majority of instances), the trachea and esophagus may be constricted and give rise to serious clinical symptoms. Wolman[15] has given an excellent description of them. Shortly after birth there seem to be difficulties in swallowing, and stridor is noticed, which may be inspiratory or expiratory, or both. Sometimes this symptom is observed only when the child cries, or exerts himself. At times there is cyanosis, in other cases this symptom is evoked only by crying. Intercostal and suprasternal contractions may be noticed. Moreover, the child often has fits of curious,

raucous coughing. The onsets and the asphyxia are very marked during the periods of feeding, when stridor mostly increases and cyanosis develops. The action of feeding, moreover, is difficult, and takes much time, as between the acts of swallowing, the child must rest in order to recover his breath. Infection of the air passages occurs frequently; it is often caused by swallowing the wrong way, ending fatally when it is not attended to. In the seven cases collected by Wolman[15] all the patients died.

The view advocated by Apley in the Royal Society of Medicine in 1949, in a discourse entitled "Congenital Anomaly of the Aortic Arch and Its Branches" would therefore seem to be somewhat unusual, at least in the form in which it was published. In the summary it is stated that "Treatment, apart from chemotherapy of superimposed lung infections, is rarely indicated, because of the tendency to spontaneous recovery, but surgical correction may be life-saving."

It is not quite clear whether this view has special reference to a certain form of vascular anomaly in this case—the double aortic arch. If this should be so, we would observe that in those cases where the anomaly was accidentally discovered at an obduction, or where they were accidentally diagnosed in adults, there had been no clinical symptoms previously. A short time ago we found this anomaly accidentally in a man, aged 69 years, who complained of vague gastric pains. He had never before had any difficulties in swallowing, and there had not been any subjective or objective symptoms of narrowing of the trachea. On the other hand, however, we know of many cases where the symptoms arose a short time after birth, and where the patient died of pneumonia. When the irregularity reveals itself in distinct symptoms in young children, we must look upon it as very grave. Potts[10] for example, treated an 8-year-old child, who had no fewer than twenty-one times been admitted to the hospital for pneumonia before being operated upon. I therefore agree with Exalto, Dicke, and Aalsmeer,[4] when they expressed the view that in babies or young children surgical therapy is necessary when the ring of blood vessels develops clinical symptoms, in order to prevent death or repeated infections of the air passages. I would, however, add that in such a case the symptoms must be really pronounced and unmistakable, for naturally there will also be cases where the symptoms are of such a mild degree that it seems possible to carry them along without an operation. When we consider the fact that Gross and Neuhauser[16] report four deaths in sixteen cases, it seems necessary to avoid the greater risk of an operation in such mild cases, where the anomaly itself will mean a very slight risk for the patient.

With the aid of a roentgen examination, a diagnosis can be made during life. Neuhauser[9] pointed out that after swallowing barium a filling defect may be observed in the posterior wall of the esophagus due to pressure of the right posterior aortic arch. The trachea proves to be constricted at the same place, which may be made visible in an ordinary roentgen photograph; if required, lipiodol may be introduced into the air passages.

It is hardly necessary to say that the only rational therapy of this irregularity consists of cutting the constricting vascular ring. The operation in itself is not difficult. As far as I have been able to ascertain it has up to now been successfully done on eighteen patients only.

In 1945 Gross[6, 7] published the first successful case, in 1946 he published a communication about a second patient cured, and from a correspondence with Potts[10] it appears that he had a third successful case, which had not yet been published. In 1947 Sweet[13] mentions two patients who were successfully operated on. In 1948 Potts[10] described a female patient who was operated on with complete success, and he also mentioned the case of another child where the cause of the constriction could not be found during the operation, as the constricting vessel lay hidden behind the esophagus; this child died some days later of suffocation. In 1950, finally, Exalto, Dicke, and Aalsmeer[4] published three cases of the same anomaly which were treated successfully.

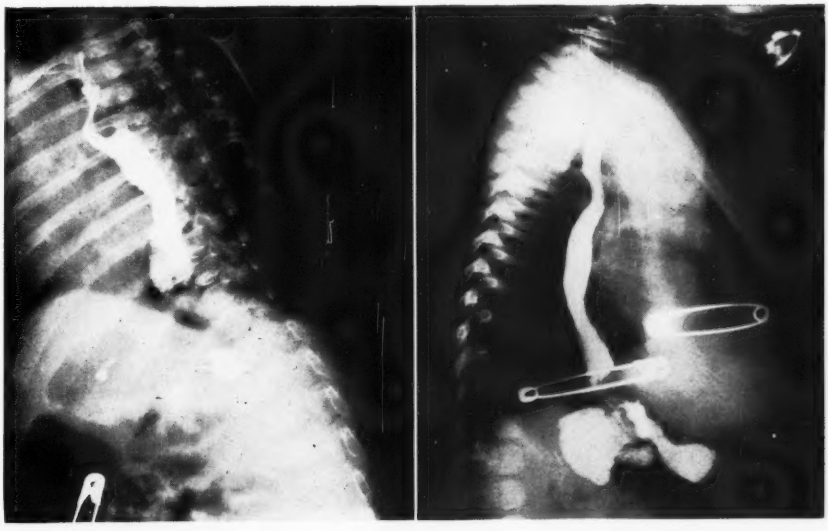

Fig. 2. Fig. 3.

Figs. 2 and 3.—Roentgenogram of esophagus after barium filling. A filling defect will be observed toward the rear, at the level of the third and fourth thoracic vertebrae.

In *Pediatrics* (January, 1951) Gross and Neuhauser[16] reported on no fewer than sixteen cases of double aortic arch which were surgically treated. The ages varied between 1 month and 2 years. In thirteen patients there was a left descending aorta; in nine of them the left (anterior) arch was the smaller of the two and was divided; in four cases the smaller right (posterior) arch was cut. In the three cases where a right descending aorta was seen, it was always the left (posterior) arch that was divided. Four of these sixteen patients died. The cases published earlier by Gross are included in this series.

I can add the following case to this short series:

A 3-month-old Belgian boy from Antwerp, born Aug. 12, 1950, the fourth child of healthy parents (weight at birth 4,000 Gm.), was admitted to the Surgical Department of the University Hospital at Utrecht on Nov. 23, 1950. From his birth onward it was noticed that the child suffered from a distinct stridor. Supraclavicular and intercostal inspiratory contractions were observed. The child cried a great deal, and he often had mucus in the air passages, causing fits of coughing. Respiration was easier when he was held erect. The act of drinking caused troubles from the first; the child drank very slowly, feeding had to be interrupted repeatedly, and he easily choked when swallowing. When he was crying, cyanosis developed. The pediatrician in Antwerp, Dr. Helderweirt, made roentgen photographs, which at first revealed only hypertrophy of the thymus. After the esophagus had been filled with barium, a considerable depression in the posterior wall of the esophagus was observed near the third and fourth thoracic vertebrae (Figs. 2 and 3); in the anteroposterior roentgenograms, too, a constriction could be seen at this site. The trachea also proved to be narrowed here (Fig. 4). An attempt to fill the trachea with lipiodol failed because it caused an onset of extreme suffocation. A diagnosis of "double aortic arch" having been made, the child was sent to us for surgical treatment.

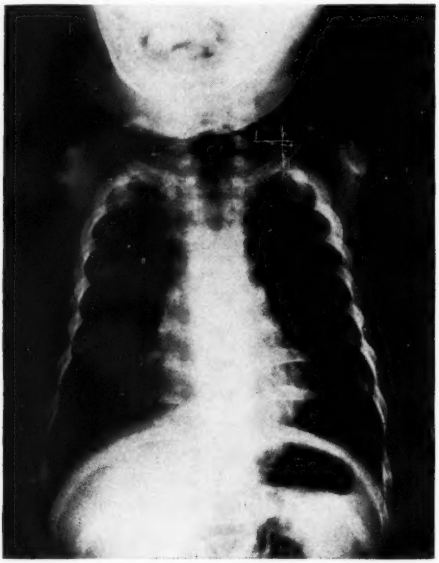

Fig. 4.—Roentgenogram of trachea. A stricture is visible just above the bifurcation.

On repeating the roentgenologic examination the above findings were fully confirmed. Considering the circumstance that symptoms of pressure on the trachea and esophagus could be observed, which are characteristic of constriction by a double aortic arch, the diagnosis seemed to be correct. The child showed symptoms of serious constriction of the two anatomic organs, and therefore the prognosis without surgical treatment was very bad; hence it was decided to proceed to an operation. The patient was somewhat more than three months old, its weight was 5,700 Gm., and its general condition was good.

The operation was performed on Dec. 4, 1950, intratracheal anesthesia of cyclopropane-oxygen being administered. The tracheal catheter number 1 could not be advanced beyond the stricture, and up to the moment when the constricting ring was cut the anesthetist found

it very difficult to make the gases pass the site of the stricture in the air passages. The patient lay on his right side, and the thoracic cavity was opened in the third intercostal space. The large thymus was at once conspicuous. There was no aortic arch on the left side, and at first the descending aorta could not be seen. Below the pleura a fairly large pulsating vessel was visible, which immediately gave the impression of being the patent ductus Botalli. The mediastinal pleura was incised between the phrenic nerve and the vagus nerve, exposing the left pulmonary artery. The blood vessel observed earlier proved to be the long ductus Botalli. After the thymus was moved aside the anatomic situation became clear (Fig. 5).

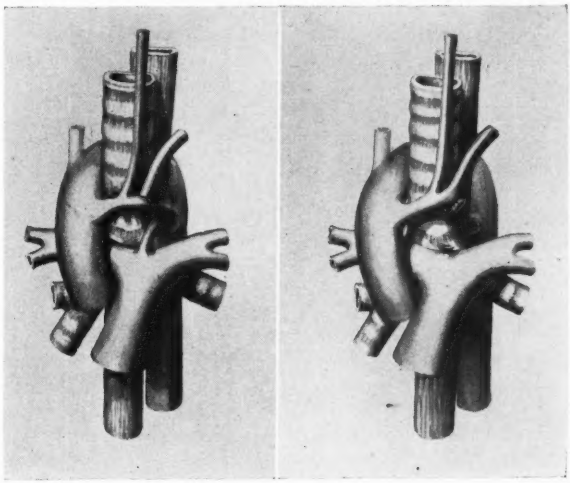

Fig. 5. Fig. 6.

Fig. 5.—Anatomic situation as revealed by the operation. There is a wide, right, posterior aortic arch, and a narrow, left, anterior arch, converging left and right behind the esophagus, forming the descending aorta, which lies somewhat farther to the right than normally. At the site where the anterior and posterior arches unite the former is slightly widened, and receives the long ductus Botalli. From the anterior arch rise the left carotid and subclavian artery; the innominate artery could not be identified, it probably sprang from the posterior arch. Esophagus and trachea are constricted by a ring of blood vessels.

Fig. 6.—Anatomic situation at the end of the operation. The ductus Botalli has been resected, the anterior aortic arch has been ligated and divided at the point of union with the posterior arch.

There appeared to be a fairly slender anterior aortic arch, from which the left subclavian artery and the left carotid arose. The posterior aortic arch was short and thick. It passed into a descending aorta, which lay slightly farther to the right than is normal, exactly in front of the vertebral column. Near the site where the anterior and the posterior aortic arch united, slightly to the left and behind the esophagus, the anterior aortic arch was somewhat wider and received the ductus Botalli. The right carotid and the subclavian artery could not be identified; these vessels probably arose on the right from the posterior aortic arch. We could see very clearly that the trachea and esophagus were constricted by this ring of blood vessels.

When the anatomic situation had become clear, the type of operation suggested itself speedily. A double ligature was applied to the ductus Botalli, and the adjacent part then resected. Next a double ligature was also applied to the anterior aortic arch at the site of the union with the posterior arch, followed by resection (Fig. 6). At that moment the esophagus and the trachea were released from their stricture. The esophagus protruded in a lateral sense, and at the same time the trachea became passable so that the catheter could be advanced beyond the stenosis. The thoracic cavity was closed again without drainage.

After the operation the child was placed in an oxygen tent. His condition was excellent, although a slight stridor remained. Feeding then involved no further difficulties. The oxygen tent was removed four days later, recovery was uneventful, and on the fourteenth day the patient was discharged. Before his departure fresh roentgenograms were made. The site of the former stenosis in the esophagus was then readily passed by the barium meal (Figs. 7 and 8).

Reports from the parents and the pediatrician at Antwerp show that the child is doing very well, and that the stridor has gradually disappeared.

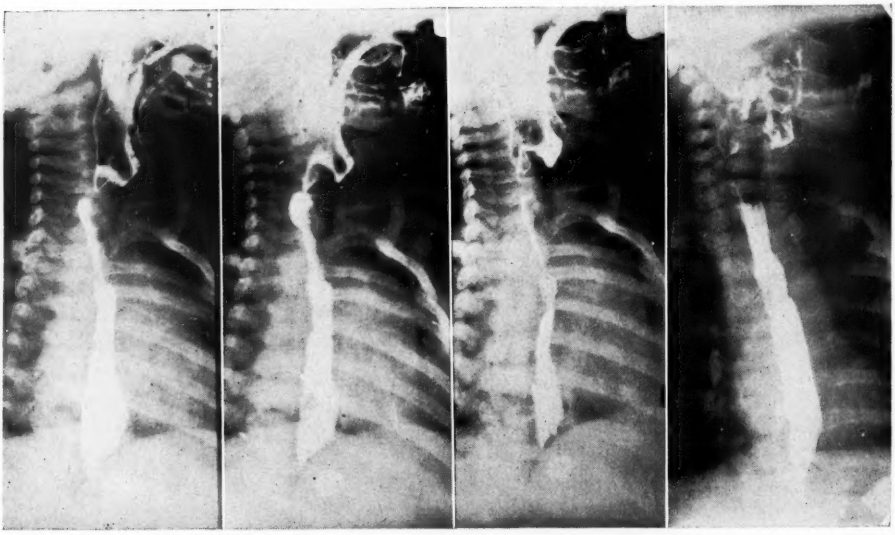

Fig. 7. Fig. 8.

Fig.. 7 and 8.—Roentgenograms of the esophagus after the operation. The stricture is no longer visible, and the barium meal passes very easily.

Exalto et al[4] point out that although the patient's condition, generally speaking, immediately shows a considerable improvement, yet there remained a slight stridor for some time in the patients they treated; it diminished slowly during the subsequent weeks, and finally vanished completely. This agrees with our experience. It would seem quite reasonable to assume (with these authors) that the stridor is caused by a softening and distortion of the cartilage rings of the trachea, which have been constricted from the third embryonal week. These irregularities will only disappear gradually.

SUMMARY

A report is given of a case of congenital constriction of trachea and esophagus, caused by a compressing double aortic arch in a child aged a little over 3 months. An operation was successful.

REFERENCES

1. Apley, J.: Proc. Royal Soc. Med. **42**: 918, 1949.
2. Arkin, A.: Wien. Arch. f. Inn. Med. **12**: 385, 1926.

3. Blincoe: Anat. Rec. 66: 505, 1936.
4. Exalto, J., Dicke, W. K., and Aalsmeer, W. C.: Arch. Chir. Neerl. 2: 170, 1950.
5. Gordon, S.: J. Pediat. 30: 428, 1947.
6. Gross, R. E.: New England J. Med. 233: 586, 1945.
7. Gross, R. E., and Ware, P. F.: Surg., Gynec. & Obst. 83: 435, 1946.
8. Malacarne: Quoted by Gross and Ware.
9. Neuhauser, E. B. D.: Am. J. Roentgenol. 56: 1, 1946.
10. Potts, W. J., Gibson, S., and Rothwell, R.: Arch. Surg. 57: 227, 1948.
11. Sagorsky, P.: Quoted by Gross and Ware.
12. Schall, L. A., and Johnson, L. G.: Am. J. Otol. Rhinol. & Laryng. 49: 1055, 1940.
13. Sweet, H., Findlay, C. W., and Reyersbach, G. C.: J. Pediat. 30: 1, 1947.
14. Welch, W. H.: Bull. Johns Hopkins Hosp. 2: 142, 1891.
15. Wolman, I. J.: J. Pediat. 14: 527, 1939.
16. Gross, R. E., and Neuhauser, E. B. D.: Compression of the Trachea or Esophagus by Vascular Anomalies, Pediatrics 7: 69, 1951.

X-RAY THERAPY IN CARCINOMA OF THE ESOPHAGUS

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CARCINOMA of the esophagus may be treated either by surgery or irradiation, the five-year cure rate from each being extremely poor. Surgery carries with it a considerable immediate mortality, even under the best conditions. It promises little more than the removal of the primary, and is usually of a mutilating nature. True carcinoma of the esophagus is almost invariably of an epidermoid type, and as such has a moderate radiosensitivity. Radiotherapy of this disease has been abandoned in many centers in favor of surgery, or at least relegated into a place of secondary importance due to the recent technical improvements in thoracic surgery and the advent of the antibiotics.

It is the purpose of this paper to show that cancer of the esophagus can be eradicated with properly planned deep x-ray therapy, at least in the primary site, and an almost normally functioning esophagus restored.

We wish to present four cases of carcinoma of the esophagus, all proved by biopsy, and treated by multifield beam-directed deep x-ray therapy,[1] on whom postmortem examination revealed no residual tumor in the esophagus or in the near-by tissue.

From January, 1946, to December, 1950, nineteen cases of cancer of the esophagus were seen. Six patients were not treated, owing to the presence of distant metastases or poor general condition. All of these patients died within four months. Another patient refused any treatment. Twelve patients were treated with deep x-ray therapy. Five of these patients, all proved epidermoid carcinomas, are living and apparently free from disease locally for periods up to fifty-two months. The remaining seven died from three to thirty-eight months after treatment.

Of the seven treated patients who died, autopsies were obtained on only four. Biopsies before treatment in each of these cases showed epidermoid carcinoma. In each autopsy the esophagus, and the surrounding tissue were carefully examined, and multiple microscopic sections were taken. No evidence of carcinoma was found in the treated area.

CASE REPORTS

Case 1.—E. K., a man aged 66 years, was admitted to the hospital in August, 1947, complaining of increasing difficulty in swallowing solids for two years. He was still able to eat very soft solids by following each mouthful with fluids. No weight loss was evident. Roentgenogram examination revealed irregularity in the lower esophagus, but the barium

From the Saskatoon Cancer Clinic.
Received for publication April 3, 1951.

passed into the stomach. Esophagoscopy showed a friable ulcer on the anterior wall two inches below the aortic arch, and one inch further down it extended around the lumen to involve the posterior wall. The scope was passed to the cardia, and for one inch above the cardia the mucosa was normal. Biopsy was epidermoid carcinoma, grade II. The patient was treated with beam-directed deep x-ray therapy. (Four hundred kv., 5 Ma, F.S.D., 80 cm., H.V.L. 4.4 mm. Cu., six fields each 15 by 6 cm. delivering a total tumor dose to the esophagus of 6,000 r in five weeks. There were twenty-five treatments, each field daily.) Barium swallow in December, 1947, revealed a smooth esophagus with slight narrowing. He was able to swallow all foods easily. In June, 1948, it again became difficult to get solids down. Roentgenograms showed no change, and on esophagoscopy no evidence of cancer could be found. Dilatation with esophageal bougies was done intermittently. Swallowing improved, and he remained well until August, 1950, when he was readmitted to the hospital. On examination the liver was enlarged, there was a large mass in the epigastrium, and a weight loss of twenty-one pounds. Roentgenograms showed that the lower end of the esophagus was deviated far to the left by a mass outside the esophagus and stomach. He died three years and two months after the treatment. At autopsy no evidence of cancer was found in the esophagus. The scarred area, where the tumor had been situated, was completely covered with epithelium, and no cancer was seen in the numerous microscopic sections. There was metastatic involvement of the gastro-hepatic and peri-aortic lymph nodes, and from hence invasion of the stomach wall. Liver, pancreas, and thirteen vertebrae were replaced by cancer.

CASE 2.—J. R., a man aged 60 years, was admitted to the hospital in October, 1947, with difficulty in swallowing everything except semi-solids and fluids for one month, and ten pounds weight loss. Roentgenogram of the chest showed a focus of old inactive tuberculosis in the left apex. On barium swallow an irregular narrowing was seen in the mid-esophagus, and on esophagoscopy a fungating ulcerated mass was seen in the middle third extending onto the right wall and the whole of the posterior wall for a length of two inches. The mass appeared to be deep in the tissue behind the esophagus and fixed. The patient was treated with beam-directed deep x-ray therapy. (Four hundred kv., 5 Ma, F.S.D. 80 cm., H.V.L. 4.4 mm. Cu. through six fields, each 6 by 15 cm., delivering a tumor dose to the esophagus of 5500r in three weeks. Each field was treated daily for fifteen treatments.) The tuberculous focus in the left apex became active after the treatment, and the patient died in the Sanatorium four months later. Autopsy revealed no residual cancer in the esophagus on multiple microscopic sections. The treated area had not yet completely re-epithelized. Bilateral fibrocaseous tuberculosis was present with cavitation in the left apex, and there was terminal pulmonary embolism. No metastatic cancer was found. It is well known that x-ray therapy will activate pulmonary tuberculosis, but in this case it was considered that the risk was justified.

CASE 3.—N. S., a man aged 63 years, was admitted in November, 1947, with pain in the retrosternal area and difficulty in swallowing food, and food sticking in the lower esophagus for four months. No weight loss. Roentgenogram revealed an irregularity in the mucosal pattern of the lower five inches of the esophagus with normal mucosa seen below the diaphragm. On esophagoscopy a fungating growth with narrowing involved the anterior and right side of the lumen in the lower five inches of the esophagus. It was impossible to pass the scope beyond the lower edge of the growth. Biopsy was epidermoid carcinoma, grade III. The patient was treated with beam-directed deep x-ray therapy. (Four hundred kv., 5 Ma, F.S.D. 80 cm., H.V.L. 4.4 mm. Cu., through six fields each 15 by 6 cm., delivering a tumor dose of 5500r in three weeks. Each field was treated daily for fifteen treatments.) The patient remained well and was able to eat all foods until April, 1948, when he developed retrosternal pain related to exertion. In December, 1948, he was admitted to the hospital in congestive failure, and did not respond to treatment. He died fifteen months after treatment. Autopsy revealed no evidence of cancer in the esophagus, although multiple microscopic sections were done, and no evidence of distant metastases was present. The patient died with congestive failure incident to cardiac infarction following coronary thrombosis.

CASE 4.—J. T., a man aged 52 years, was admitted January, 1950, complaining of increasing difficulty in swallowing solids for eight months and taking liquids only. He had a rib resection for empyema and a pericarditis in 1918, but no other illness. Barium swallow showed an obstruction at the level of the aortic arch with moderate dilatation above. The upper esophagus and trachea were displaced forward by an abnormal soft tissue mass which extended up toward the neck. A barium tract one inch long was observed to extend posteriorly from the esophagus into the mass. On esophagoscopy there was an extensive fungating tumor at the junction of the upper and middle thirds. It was not possible to find the lumen. Biopsy was reported as squamous cell carcinoma. The patient was given a course of beam-directed deep x-ray therapy using the Betatron (22 M.E.V., F.S.D. 105 cm., three fields, each 15 by 6 cm. delivering a tumor dose of 6000 equivalent roentgens in three weeks. Each field was treated daily for fifteen treatments.) One week after treatment was finished he had less difficulty in swallowing and barium showed the narrowing in the lumen to be less irregular. Chest roentgenograms showed the upper mediastinal mass to be smaller. The patient returned home to New Zealand and died three months later. On postmortem examination there was a bridge of normal mucosa on the anterior wall of the esophagus. The posterior wall was ulcerated and attached to the bodies of the seventh cervical and first thoracic vertebrae. The bone was exposed, and the anterior parts of the bodies formed the posterior wall of a ragged retroesophageal abscess cavity. Sections from the base and edges of the abscess were free from tumor growth. No evidence of residual cancer was found in the lymph nodes and no distant metastases.

CONCLUSIONS

It is shown that cancer of the esophagus can be eradicated by properly planned beam-directed deep x-ray therapy. Four cases with clinical history and postmortem findings are reported.

The autopsies in Cases 1, 2, and 3 were performed by Dr. J. W. Adams, Pathologist, Saskatoon City Hospital, and in Case 4 by Dr. P. P. Lynch, Pathologist, Wellington, New Zealand.

REFERENCE

1. Paterson, R.: The Treatment of Malignant Disease by X-rays and Radium, Arnold and Company, London, 1947.